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The Effects of Sauna Bathing on Health Markers in Middle Aged Males and Females

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The Effects of Sauna Bathing on Health Markers in Middle Aged Males and Females

by

Michelle Dotzert

A Thesis
Submitted to the Faculty of Graduate Studies
through Kinesiology
in Partial Fulfillment of the Requirements for
the Degree of Master of Human Kinetics at the
University of Windsor

Windsor, Ontario, Canada

2012

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The Effects of Sauna Bathing on Health Markers in Middle Aged Males and Females

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Author's Declaration of Originality

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Abstract

Sauna bathing improves heart rate variability (HRV), resting blood pressure (BP), fasting plasma glucose (FPG) and body composition in patients with chronic heart failure (CHF) and diabetes, and benefits depressed patients. Sauna bathing elicits similar physiological responses to exercise, including increases in core temperature, sweating, heart rate and skin blood flow. Exercise training decreases cardiovascular and metabolic disease morbidity and mortality rates. Despite these benefits, only 50.9% of Windsor-Essex County is moderately physically active. Further, reduced aerobic fitness, BP, FPG, body composition, etc. occur with age. As such, it is of interest to examine whether sauna bathing as a form of passive heating prevents the onset of cardiovascular and metabolic disease by improving health markers in middle aged adults. To examine this question, we recruited 5 healthy, sedentary to moderately active middle-aged participants to undergo sauna bathing 5 times per week for 2 weeks. The sauna intervention consisted of a 15 minute dry sauna exposure at approximately 60°C followed by 30 minutes of covered rest. Resting HR and BP, HRV, body composition, blood lipids (TC, HDL, LDL, TRG), FPG, hematocrit (HCT) and state trait anxiety (STAI) were measured at baseline, following two weeks of normal activity and following two weeks of daily sauna bathing. Tympanic temperature was measured during each sauna exposure and showed a significant increase following the 15 minute sauna bath ($p < .001$). Resting HR, BP, HRV, body composition, STAI, blood lipids, and FPG were unchanged following two weeks of sauna use ($p > .05$). HCT increased following two weeks of sauna use ($p < .05$). These results suggest that daily dry sauna bathing with a concomitant 1.9 ± 0.6 °C tympanic temperature increase does not improve markers of cardiovascular, metabolic and psychological health among generally healthy middle-aged adults.

Dedication

I would like to dedicate this work to my family and friends. Mom, Dad, and James, thank you for everything you do. I wouldn't be where I am today without your ongoing support.

To Kali and Kevin, you are two of the most intelligent and spirited people I've met. I'm so fortunate to have worked with you.

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Chapter 1-Literature Review

1.0 The origins of thermal therapy and saunas

Originating in Finland, the sauna bath is now popular worldwide. What was first described by the Greek historian Herodotus in 440 BC as “three pieces of wood leaning against each other” with “red-hot stones...in the middle” (Herodotus & Bähr, 1848) has become common practice in northern Russia, Finland, Norway, and Iceland while similar customs have been practiced among Eskimo and American Indian tribes of Alaska and Canada as well as parts of South America (Lopatin, 1960). While the methods of passively heating the body are different, they all involve raising core temperature for brief periods. For example, in Central Asia, the Turkish bath involves moving between warm and hot rooms, followed by immersion in cool water. Heating is also a component of the Arabian bath (*hammam*), although warm stone platforms take the place of steam or dry heat. Unlike these practices, which focus on cleansing the body, *kashim* baths are common among Alaskan Eskimos and involve dry heat from fire followed by a brief period of snow or ice exposure (Lopatin, 1960).

Early sauna bathing was considered a spiritual process, however, the popularity of the modern sauna is more closely tied to the health and fitness industry. Numerous claims associate regular sauna use with weight loss, sweating “toxins” out of the body, as well as greater feelings of well being and relaxation (Biro, Masuda, Kihara, & Tei, 2003; Cecchini & LoPresti, 2007; Masuda, Nakazato, Kihara, Minagoe, & Tei, 2005). While evidence of these health effects is limited, more recent literature focuses on the potential

cardiovascular benefits of passive heating (Imamura et al., 2001; Kihara et al., 2004, 2009).

1.1 Exercise is a physiological stress

Chronic stress, both physiological and psychological, is associated with a variety of negative health outcomes (Thoits, 2010). However, chronic physical activity, involving shifts away from internal homeostatic set points and consequently a stress to the physiological system, leads to improvements in human physiological functioning and psychological well-being. The stresses resulting from exercise include increased oxygen demand, elevated body temperature (Radomski, Cross, & Buguet, 1998) tissue damage (Powers, 2008) reduced pH (increased acidity) and a host of metabolic stressors such as increased reactive oxygen and nitrogen species (Powers, 2008) and an increase in cytokines (Vassilakopoulos et al., 2003) among others. Further, as exercise intensity and/or duration increases, these stresses are augmented (Fisher-Wellman & Bloomer, 2009). Nonetheless, the beneficial effects of chronic exercise undoubtedly occur because the body adapts to these stresses. These adaptations include changes in fuel mobilization (Sherman, 1995), changes in muscle fibre type (Pette & Staron, 2001; Röckl, Witczak, & Goodyear, 2008), more efficient oxygen extraction (Cooper, Beaver, Cooper, & Wasserman, 1992), improved vascular control (Higashi et al., 1999; Rowell, 1974) as well as increased sweat production (Ichinose, Inoue, Hirata, Shamsuddin, & Kondo, 2009; Rowell, 1984). This resistance is conferred through improved cardiorespiratory fitness, and changes in markers of metabolic and cardiovascular disease. With regular exercise, reductions in type II diabetes (Sigal, 2006), hypertension (Chobanian et al., 2003; Gupta & Guptha, 2010), coronary artery disease (CAD) and CAD symptoms

(Thompson & Lim, 2003) are observed, and not surprisingly then, so too are reductions in premature mortality (Blair et al., 1996). Furthermore, exercise enhances psychological well-being (Taylor, Sallis, & Needle, 1985) and may provide therapeutic benefits for those with depressive disorders (Babyak et al., 2000). Therefore, exercise as a physiological stress produces positive health outcomes.

Nonetheless, many individuals do not make physical activity a part of their daily lives. In Windsor-Essex County, only 50.9% of the population is moderately active or active (Statistics Canada Health Profile, 2011). The current physical activity recommendations for adults 18-64 are 150 minutes of moderate to vigorous aerobic activity each week (Canadian Society for Exercise Physiology, 2012). However, common reasons that individuals provide for not meeting physical activity requirements are insufficient time and fatigue (Brownson, Baker, Housemann, Brennan, & Bacak, 2001). Moreover, some negatives associated with exercise are the risk of injury (e.g. sprains, strains, falls, etc.) and the physical demanding nature of the activity. In contrast, passive heating represents a cardiovascular strain, but eliminates some of these negatives and consequently, the role of heat therapy and the subsequent cardioprotective and/or disease-protective effects may provide a viable alternative to traditional exercise training and rehabilitation.

1.2 Exposure to heat is a physiological stress

Passive heating stimulates a physiological response similar to that of exercise, whereby the body must work to resist a shift away from homeostasis and ensure survival. Chronic exposure to high ambient temperatures without appropriate rest, fluid and

electrolyte replacement is detrimental to human health, potentially resulting in heat illness, heat stroke or death. The physiological stress of extreme temperatures is most dangerous for young children (due to small body size) and the elderly, who are often mildly dehydrated or have an underlying condition exacerbated by heat. In an effort to minimize these events, Environment Canada issues heat alerts and advisories when Canadian summer temperatures exceed 30°C.

Depending on the severity of heat stress, heat exhaustion or heat stroke may occur. Sweating, headache, nausea, vomiting, and dizziness characterize heat exhaustion while symptoms of heat stroke include core temperatures above 40°C and central nervous system dysfunction (Canadian Centre for Occupational Health and Safety, 2008). Heat illness has been reported at various sport and exercise events, such as the 2007 Chicago Marathon. At that event, one fatality and forty-nine hospitalizations were documented due to high temperatures and humidity (Rousseau, 2007).

To account for the stress of elevated body temperature, cardiovascular adaptations, many of which are similar to the adaptations observed with exercise, occur. The body must dissipate heat to prevent the denaturation and dysfunction of proteins vital to physiological and metabolic processes. To do so, heart rate increases approximately ten beats per minute for each degree (1°C) of body temperature increase (Clark & Edholm, 1985). This elevation aids the process of evaporative cooling, where increased skin blood flow couples with eccrine (the major sweat glands found all over the body) sweating to dissipate heat (Gagnon, 2009). For example, when resting heart rate and oral temperature were compared to values following an 80°C dry sauna exposure, an approximate 1°C oral

temperature increase elicited a heart rate increase from 71 ± 12 beats per minute to 96 ± 18 beats per minute (Kukkonen-Harjula et al., 1989). Similar heart rate changes have been observed following an 80°C sauna exposure (Leppäluoto, Tuominen, Väänänen, Karpakka, & Vuori, 1986).

Heart rate comprises a component of cardiac output (Q), where Q is a function of heart rate (HR) multiplied by stroke volume (SV), and outlined by the following equation:

$$\text{EQ.1} \qquad \qquad \qquad Q \text{ (mL/min)} = \text{HR (bpm)} \times \text{SV (mL)}.$$

As such, heat stress is accompanied by greater cardiac output. In fact, heat exposure can increase skin blood flow from 300 mL/min^{-1} to 7500 mL/min^{-1} , as flow is directed away from non-essential organs. Furthermore, increased cutaneous flow causes a decrease in central blood volume, and a 3-5 mmHG reduction in central venous pressure (Minson, Wladkowski, Cardell, Pawelczyk, & Kenney, 1998). These reductions, coupled with unchanged or slightly elevated stroke volumes lead to greater myocardial contractility (Brothers et al., 2009; Rowell, 1984). As stroke volume is not significantly changed during heat stress, increased cardiac output is the result of elevated HR (Minson et al., 1998; Wilson et al., 2007) occurring with core temperature elevations of 1°C to 1.5°C (Fan et al., 2008).

In addition to cardiovascular changes, increased skin blood flow couples with eccrine sweat production to aid in thermoregulation. When ambient temperatures are greater than

skin temperatures, increased skin blood flow permits heat from the blood to combine with water in sweat to undergo a phase change and form a gas, thus dissipating heat from the body (Shibasaki & Crandall, 2010). To further aid in thermoregulation, sweat production occurs. The eccrine sweat response is initiated centrally by the hypothalamus and aids in evaporative cooling (Morrison & Nakamura, 2011).

Furthermore, as with exercise, the effects of whole body thermal stress are countered with ventilation changes. As early as 1905, a study of coal mining conditions revealed the effects of high ambient temperatures on ventilation (Haldane, 1905). Haldane entered the mines and experienced these effects first hand, and reported “breathing deeper and more frequent” at an oral temperature of 102.6°F (Haldane, 1905). Later animal work also demonstrated ventilatory changes during heating using canine carotid bodies perfused with heated blood (Bernthal, 1939). The carotid bodies are located on the wall of the left and right common carotid arteries, and, along with the aortic bodies on the wall of the aortic arch, are sensitive to oxygen, carbon dioxide and pH changes in the blood. It has also been suggested these bodies are temperature sensitive (Bernthal, 1939; Gallego, Eyzaguirre, & Monti-Bloch, 1979; Loyola et al., 1991). When change is detected, ventilation is altered to restore homeostasis. In the case of elevated blood temperature, ventilation increases to aid in thermoregulation through the exhalation of warm air. These effects have been observed in humans exposed to hyperthermic conditions, and in instances when esophageal temperatures increase up to +2°C (Curtis, Walsh, & White, 2007; Fan et al., 2008).

Heating may also influence oxygen extraction, especially by essential organs (González-Alonso et al., 2004). Given that peripheral blood flow increases during heat stress, it is crucial for the brain to receive an adequate oxygen supply in order to maintain essential metabolic processes. Fan et al. (2008) noted that despite changes to peripheral blood flow, cerebral oxygenation remained unchanged during passive heating. Similarly, hyperthermic exercise trials elevating core temperature to $39.5 \pm 0.2^{\circ}\text{C}$ result in a seven percent increase in cerebral oxygen uptake (Nybo, Secher, & Nielsen, 2002). Greater oxygen extraction contributes to larger arterial-venous oxygen differences $((a-v)\text{O}_{2\text{diff}})$ and given the intimate nature between oxygen consumption and the cardiovascular system, can be included in what is known as the Fick equation:

$$\text{EQ.2} \quad \text{VO}_2 = Q \times (a-v)\text{O}_2 \text{ diff.}$$

This relationship between cardiac output (Q), $(a-v)\text{O}_2$ difference and oxygen consumption (VO_2) also exists during exercise as skeletal muscle oxygen consumption increases, and is important to the understanding of why the cardiovascular system adapts to both exercise and passive heating.

1.3 Mechanisms of physiological responses to heat

The mechanisms behind these physiological responses to heat are not entirely known, but it has been shown that as environmental temperature increases, warmth receptors in the skin are activated (Morrison & Nakamura, 2011). These neurons synapse in the dorsal horn of the spinal cord and the signal travels to the brain, or more specifically, the medial preoptic area and median preoptic nucleus of the hypothalamus (Morrison & Nakamura, 2011). Neurons from these regions inhibit excitatory pathways that drive

sympathetic thermoregulatory effectors (pathways), causing a decline in thermogenesis and an increase in cutaneous vasodilation (Morrison & Nakamura, 2011). The preoptic hypothalamus also increases eccrine sweating, as efferents (i.e. neuronal output) from this brain centre innervate sweat glands (Shibasaki & Crandall, 2010). Eccrine sweat glands are distributed all over the body, but are highly concentrated on volar surfaces, such as the palms of the hands and soles of the feet (Henrikson, Kaye, & Mazurkiewicz, 1997).

The secretory portion of the sweat gland has a coil structure, consists of clear and dark cells, which contain secretory vacuoles and aquaporins (i.e. cellular membrane spanning water channels) for fluid excretion, and is located at the dermo-subcutaneous junction (Munger, Van Tassel, & LaFleur, 2007; Nejsum et al., 2002). The isotonic fluid (having the same salt concentration as blood) produced in the secretory portion travels through the duct where it is released onto the skin surface.

Coupled with vasodilation, sweating aids in thermoregulation through evaporative cooling. Nerve endings on the skin containing Transient Receptor Potential Vanilloid (TRPV) channels that detect changes in temperature, and this sensory information is sent to the POAH (preoptic area of the hypothalamus) along with input from central thermoreceptors in the hypothalamus (Morrison & Nakamura, 2011). Sympathetic efferents innervating the smooth muscle surrounding arterioles, stimulate relaxation and consequently dilation to increase blood flow (Shibasaki & Crandall, 2010).

Further, when CO_2 crosses the blood-brain-barrier, hydrogen ions (H^+) are formed. Central chemoreceptors are sensitive to changes in $[\text{H}^+]$, and initiate increased breathing

depth and frequency to buffer these byproducts. Curtis et al. (2007) suggested that these chemoreceptors may also be temperature sensitive, accounting for the alterations in ventilation during passive heating. Interestingly, Bernthal (1939) observed significant ventilatory increases when they perfused isolated canine carotid bodies with 45°C blood. In humans, greater ventilation occurs in hyperthermic compared to normothermic low-intensity euoxic exercise (i.e. exercise in normal oxygen conditions) (Chu, Jay, & White, 2007). An important note about the central control regulation of body temperature is that it is predominantly the blood temperature entering the brain (carotids) or surrounding the hypothalamus that signal body temperature. For this reason, measuring temperature of the blood passing by the hypothalamus (e.g. using a tympanic thermometer) has been suggested to be optimal when studying the impacts of heat exposure (Lee et al., 2011).

Overall, in response to heat, there is an increase in blood flow to the skin, resulting in more work for the cardiovascular system to meet the blood flow (and oxygen) demands of vital organs such as the brain. It is likely that many of the health challenges, but also chronic benefits associated with passive heating are a result of this increased demand on the cardiovascular system.

1.4 Mechanisms of heart rate variability (HRV) improvement

To ensure survival, an organism must be able to respond to its environment. The ability to outrun a predator, for example, relies on the “fight-or-flight” response characterized by changes in heart rate, blood flow to working muscle, pupil dilation and fuel mobilization. While we are usually no longer faced with these types of threats to survival, a healthy human heart must be able to respond to environmental changes, both

psychological (e.g. interpersonal and work stressors) and physiological (e.g. heat, illness and exercise).

The sinoatrial (SA) node is considered the heart's pacemaker, given its ability to spontaneously contract without nervous input approximately 100 times per minute. The autonomic (involuntary) nervous system influences heart rate via sympathetic (SNS) and parasympathetic (PNS) activity. The SNS, associated with fight or flight, is normally considered the accelerator, while the PNS, associated with rest and digest, is normally considered the brakes of the system. Through predominantly PNS activity acting through the vagus nerve innervating the SA node, resting heart rate is maintained between 60 and 70 beats per minute on average. Parasympathetic terminals in the SA node slow depolarization through the release of acetylcholine. Acetylcholine is a small neurotransmitter (chemical messenger) that binds to and activates membrane potassium (K^+) channels, thereby increasing K^+ permeability. This results in cardiomyocyte hyperpolarization (i.e. further away from the threshold necessary for depolarization and the stimulus for myocardial contraction) and a decreased rate of SA node firing (Berntson et al., 1997). The SNS also acts through the vagus nerve to modulate heart rate. Sympathetic terminals speed SA node rhythm by releasing Norepinephrine (a neurotransmitter), which acts on β_1 receptors to stimulate a signal cascade within the cell (Berntson et al., 1997). Further SNS regulation occurs with the release of adrenomedullary catecholamines (hormones involved in the stress response) (Berntson et al., 1997).

Heart rate variability (HRV) is a measure of SNS and PNS regulation of heart rate, where greater variability is indicative of a greater capacity to respond to a changing environment. Lower variability due to reduced PNS control is a predictor of cardiovascular morbidity and mortality (Routledge, Campbell, McFetridge-Durdle, & Bacon, 2010). Short-term recordings of HRV ranging from 2-5 minutes are used to produce three spectral components: very low frequency (VLF), low frequency (LF) and high frequency (HF) (Camm et al., 1996). The HF component consists of vagal activity, while the LF component is a combination of SNS and PNS activity. The LF/HF ratio represents sympatho/vagal balance, and therefore a lower ratio is indicative of greater vagal activity

To measure HRV, the N-N (normal-to-normal) intervals are calculated from electrocardiogram (ECG) recordings of the heart's electrical activity. N-N intervals are the period between adjacent QRS complexes resulting from sinus node depolarization (Reed, Robertson, & Addison, 2005) (see Figure 1). It should be noted that N-N interval refers to R-R

Figure 1.

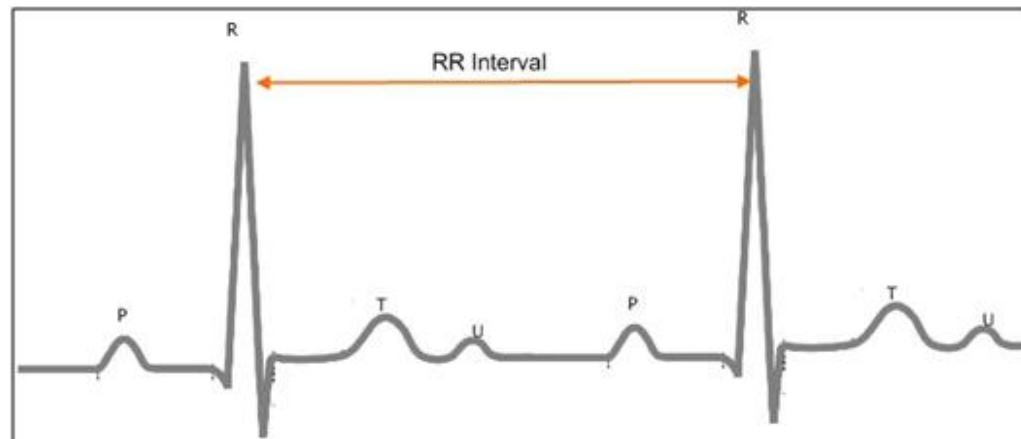


Figure 1. A normal, one-cycle ECG signal measured in the standard lead V6.

The RR interval is the time between two successive R waves. P represents atrial depolarization followed by a delay as the electrical activity passes through the AV node. The QRS complex represents ventricular depolarization, and the T wave represents ventricular repolarization.

intervals that have been processed as “normal”. Greater standard deviation of N-N intervals (SDNN) is associated with better autonomic regulation, whereas reduced SDNN has implications in chronic heart failure (Hooper, 1999), and may couple with the inflammatory process to be a cause of the formation of atherosclerotic plaques (Sajadieh et al., 2004). Moreover, HRV is altered in many disease states. For instance, post-myocardial infarction measures of vagal activity and overall HRV are lower (Cerati & Schwartz, 1991). Loss of vagus activation during sleep has also been reported, and may provide insight into the occurrence of nocturnal sudden death (Vanoli et al., 1995; Curtis et al., 2007; Fan et al., 2008). Furthermore, decreased HRV index in post-MI patients prior to discharge was found to be an independent risk factor for left ventricle dilation at follow-up (Dambrink et al., 1994).

In chronic heart failure (CHF) patients, reduced HRV is indicative of reduced PNS activity and may have prognostic value for the increased risk of sudden death (Galinier et al., 2000; Nolan et al., 1992). In a 2000 study, Galinier et al. reported an absent LF component in patients with severe CHF. Given that the LF component is considered sympathetic control, it seems counterintuitive that it is missing, as increased sympathetic drive correlates with the increased heart rate associated with heart failure. A possible explanation is that in severe CHF all mechanisms are active in order to compensate for the pathophysiology, and as a result there is no reserve to maintain variability (Galinier et al., 2000). Nonetheless, time and frequency domain analysis of HRV has been shown to provide insight into autonomic cardiovascular modulation among hypertensives (Parati, Saul, Di Rienzo, & Mancia, 1995). Both males and females

with systemic hypertension have reduced HRV, and lower HRV among normotensive males is associated with a greater risk of developing hypertension (Singh et al., 1998).

In addition to CVD, HRV declines with aging. VLF, LF, and HF powers as well as fractal dimensions have been shown to decline with age during both awake and sleeping measures (Yeragani, Sobolewski, Kay, Jampala, & Igel, 1997). Furthermore, Choi et al (2006) report an overall sympathetic activity increase and a concomitant parasympathetic decline with aging that may account for the increased rate of CVD at this time.

Various studies demonstrate HRV improvements with exercise. Cardiac rehabilitation exercise interventions improve HRV measures post-myocardial infarction. Leitch et al. (1997) reported improvements in SDNN, Total Power, HF power and LF power following exercise training, and Oya et al. (1999) observed improved HF power following aerobic training. Exercise is also associated with HRV improvements among chronic heart failure (CHF) patients (Pietilä et al., 2002). Following six months of exercise training, greater HF power and total R-R variability have been reported (Pietilä et al., 2002). Furthermore, individuals with coronary artery disease (CAD) showed improved R-R variability following two weeks of training (Iellamo, Legramante, Massaro, Raimondi, & Galante, 2000).

Physical activity also improves HRV in non-clinical populations. In canine models, aerobic exercise may reverse autonomic neural remodeling due to myocardial infarction (Billman, 2009). In humans, SDNN (ms) (the Standard Deviation of all normal R-R intervals) is significantly linked to VO_{2max} , suggesting greater aerobic fitness is

associated with greater HRV (Aslani, Aslani, Kheirkhah, & Sobhani, 2011). Fitter individuals also demonstrate less pronounced reductions in HRV during mental stress (Hamer & Steptoe, 2007). These studies would indicate a beneficial effect of exercise on autonomic control.

The response to thermal stress, very similar to the physiological response to exercise stress, also influences HRV. Acutely, even mild heat stress (35°C for 30 minutes) is sufficient to influence components of HRV, and corresponds to a reduced HF component and increased LF/HF ratio within minutes of heat exposure (Yamamoto, Iwamoto, Inoue, & Harada, 2007). Lowered HF components, and elevated LF/HF ratios indicate a reduction in vagal activity and an increase in sympathetic drive (Crandall, Zhang, & Levine, 2000; Kinugasa & Hirayanagi, 1999). These changes suggest a reduction in vagal tone (PNS withdrawal). Nagasawa et al. (2001) examined the effects of a 40°C hot water bath on HRV in young (27.4 ± 9.6 years) and older (74.6 ± 10.7 years) participants. The results provide insight into the potential dangers of whole body heating within an older population, as the HF component was significantly suppressed within this group, but unaffected in the younger group. Thus hypotensive syncope (loss of consciousness due to low blood pressure) may result from the reduction in sympathetic tone detected during immersion (Nagasawa et al., 2001). Increased sympathetic activity during passive heating (Crandall et al., 2000; Kinugasa & Hirayanagi, 1999; Nagasawa et al., 2001; Yamamoto et al., 2007) counters the stress of rising core temperature. The sympathetic response includes accelerated heart rate, increased sweating and ventilation. It is possible that these acute sympathetic increases during heat stress improve HRV, leading to enhanced PNS activity when the stressor is removed. For example, Kihara et

al. (2004) examined the effects of sauna bathing on chronic heart failure patients. Following two weeks of daily 15 minute far infrared-ray sauna exposure, patients showed improvements in SDNN (ms) (an indication of improved HRV).

The mechanisms underlying these improvements are not fully understood, however, Angiotensin II is thought to contribute (Buch, Coote, & Townend, 2002; Townend, al-Ani, West, Littler, & Coote, 1995). Support for this theory includes the lowered plasma renin levels in athletes compared to sedentary populations (Fagard et al., 1985). Renin converts Angiotensinogen to Angiotensin I, which is subsequently converted to Angiotensin II by Angiotensin Converting Enzyme (ACE) in the lungs. Angiotensin II inhibits cardiac vagal activity (Townend et al., 1995). In an endurance trained individual, lower plasma renin may contribute to less Angiotensin II and therefore, less inhibition of vagal activity.

It is also suggested that nitric oxide is involved in exercise-induced improvements in HRV. With respect to central modulation of vagal activity, NO has been shown to increase the activity of central vagal motoneurons (Chowdhary & Townend, 1999). It also modulates an indirect vagal inhibition of sympathetic cardiac responses (Chowdhary & Townend, 1999). Research on these effects is limited, and further studies on passive heating are required to determine how temperature influences autonomic regulation.

1.5 Heat therapy and vascular changes

Studies examining the effects of extreme temperatures (Hall, Buettner, Matthes, & Gisolfi, 1994; Harris, Blackstone, Ju, Venema, & Venema, 2003) as well as more conservative sauna therapies (Ikeda et al., 2001; Ikeda et al., 2005; Miyata & Tei, 2010)

show that heat therapy provides many of the beneficial vascular changes associated with traditional exercise.

Endothelial nitric oxide synthase (eNOS) generates nitric oxide (NO) in response to various stimuli (Fleming & Busse, 2003), including oxidative stress and more importantly, shear stress (Kojda & Hambrecht, 2005). These stressors stimulate eNOS to synthesize NO from L-arginine (Ayajiki, Kindermann, Hecker, Fleming, & Busse, 1996; Corson et al., 1996; Palmer, Rees, Ashton, & Moncada, 1988) to relax vascular smooth muscle. Nitric oxide is anti-atherogenic through vasodilation, the inhibition of platelet aggregation, smooth muscle cell proliferation and leukocyte adhesion to endothelial cells (Knowles & Moncada, 1994). Similar to exercise, passive heating increases heart rate and blood flow, ultimately exerting greater shearing force on the vasculature (Tschakovsky, 2010). In support of this, 24 hours after bovine endothelial cells are exposed to 42°C heat shock, eNOS is significantly increased (Harris et al., 2003). Furthermore, maximal bradykinin-stimulated NO release is improved and the increased rate of conversion from L-[¹⁴C] arginine to L-[¹⁴C] citrulline indicates enhanced eNOS activity (Harris et al., 2003). Interestingly, more severe heat shock (45°C) is not associated with increased eNOS expression or NO release (Harris et al., 2003). Another animal study examining the effects of hyperthermia (>39°C elevation of rectal temperature) on splanchnic circulation in a rat model shows a peak in local NO 1 hour after heat exposure (Hall et al., 1994).

The role of sauna therapy in vascular remodeling has also been examined (Sobajima et al., 2011). After myocardial infarction (MI) was induced in rats, they were

placed in a dry sauna at 41°C for 15 minutes then 34°C for 20 minutes daily for 4 weeks. Rats in the sauna group showed reduced left ventricular end-diastolic and end-systolic dimensions as well as reduced MI-induced increases in left ventricle end-diastolic pressure (Sobajima et al., 2011). Furthermore, decreases in vascular density in the non-infarcted myocardium were attenuated in the treatment group. Results indicate sauna therapy reduced cardiac remodeling after MI in rats (Sobajima et al., 2011).

Although heat shock may ultimately improve eNOS and NO activities, exposure to extreme temperatures is dangerous to human health. Nonetheless, animal studies conducted using exposure to lower temperatures also yield promising results (Ikeda et al., 2001; Ikeda et al., 2005). When Syrian golden hamsters are exposed to daily heating (39°C for 15 min followed by 30°C for 20 min) over 4 weeks, aortic eNOS is significantly increased (Ikeda et al., 2001). Furthermore, when the same treatment is applied to cardiomyopathic hamsters, a significant increase in aortic eNOS mRNA is reported (Ikeda et al., 2005).

1.6 The effects of estrogen on HRV

The steroid hormone estrogen has many cardioprotective effects. It is involved in vascular smooth muscle contractility, proliferation, matrix formation and composition (Farhat, Lavigne, & Ramwell, 1996), and potentiates NO activity on vascular smooth muscle (Gilligan, Badar, Panza, Quyyumi, & Cannon, 1994). It is important to note that these effects may be age-dependent, with benefits decreasing as time post-menopause increases (Sherwood et al., 2007).

Estrogen levels vary throughout the course of the menstrual cycle, with an estrogen spike in the late follicular phase prior to ovulation and a decline in the luteal phase. Estrogen mediates certain neuronal activity, including that which influences heart rate, blood pressure and sleep (Miller & Duckles, 2008). As such, it is important to consider menstrual cycle phase in the measurement of HRV. HF power generally decreases in the luteal phase, while LF power and the LF:HF ratio tend to increase at this time (Bai, Li, Zhou, & Li, 2009; Baker, Colrain, & Trinder, 2008; Leitch et al., 1997; Sato, Miyake, Akatsu, & Kumashiro, 1995)

1.7 Hypertension and sauna therapy

Hypertension, defined as systolic blood pressure consistently over 140 mmHg and diastolic blood pressure consistently over 90 mmHg (Hypertension Canada 2011, www.hypertension.ca), is associated with increased risk of stroke and myocardial infarction (Heart and Stroke Foundation), aneurysm, atherosclerosis, and heart failure (Hypertension Canada 2011, www.hypertension.ca). It arises from defects in endothelium-mediated vascular relaxation (Panza, 1997). Close to 19% of Canadian adults have elevated blood pressure (Wilkins et al., 2010), placing them at greater risk of these complications. This condition is particularly dangerous for females, as those with high blood pressure are 3.5 times more likely to develop heart disease than those with normal blood pressure (Corrao, Becker, Ockene, & Hamilton, 1990). An additional 20% of the Canadian population is considered pre-hypertensive (Wilkins et al., 2010), with elevated systolic blood pressure between 120-139 mmHg or diastolic blood pressure between 80-90 mmHg. Pre-hypertensives are at greater risk of developing hypertension (Chobanian et al., 2003), however, health-promoting lifestyle modifications such as diet

and exercise are effective in reducing the risk of developing cardiovascular disease (Chobanian et al., 2003). In fact, the ACSM recommends 30-60 minutes of continuous or intermittent moderate-intensity aerobic exercise on most (or all) days of the week, and resistance exercise 2 to 3 times per week (ACSM's Guidelines for Exercise Testing and Prescription, 8th Ed.). An acute bout of aerobic exercise is associated with a 5-7 mmHg decline in blood pressure, with effects lasting up to 22 hours after an endurance session (Pescatello et al., 2004). Furthermore, the greatest blood pressure declines are seen in those with the highest resting values (Pescatello et al., 2004). However, despite the effectiveness of exercise in the prevention and management of hypertension, adherence is problematic. Uzun et al., (2009) followed a group of 150 hypertension patients for 1 year, and found a mere 31% adherence to the recommended exercise program. Therefore, other prevention strategies and therapeutic interventions should be evaluated for efficacy and adherence. In particular, recent research also suggests sauna therapy as an effective approach to improving blood pressure and reducing the risk of hypertension (Giannetti et al., 1999; Imamura et al., 2001; Kihara et al., 2002; Yamaoka et al., 2004). Thus, heat therapy may be a less time consuming and less strenuous intervention or adjunct therapy to traditional methods.

1.8 Heat therapy and heart failure

In addition to animal studies, more recent research has examined the effects of heat therapy for patients with chronic heart failure (CHF) and coronary risk factors (Basford et al., 2009; Imamura et al., 2001; Kihara et al., 2002). Factors such as smoking,

diabetes mellitus, hypercholesterolemia and hypertension are coronary risk factors as they are associated with vascular damage and plaque formation. Repeated thermal therapy has been shown to improve endothelial function in patients with one or more of these risk factors (Imamura et al., 2001). 2 weeks of daily sauna exposure (15 minutes at 60°C followed by 30 minutes of rest with blankets) significantly increased percent flow-mediated dilation in this group (Imamura et al., 2001). These benefits extend to the CHF population as well. A 2004 study explored the effects of sauna treatment on patients with New York Heart Association functional class II or III CHF and at least 200 premature ventricular contractions every 24 hours. Participants underwent sauna therapy (15 minutes at 60°C followed by 30 minutes of rest with blankets) 5 times per week over 2 weeks. Results indicate increased heart rate variability, and a reduction in concentration of plasma brain natriuretic peptide (a substance secreted due to excessive cardiomyocyte stretching concomitant with heart failure) (Kihara et al., 2004).

Additional research on the long-term effects of sauna therapy for patients with CHF also shows positive outcomes (Kihara et al., 2009). 64 patients with New York Heart Association class III and IV heart failure were treated 5 times per week during admission and twice weekly after discharge with the same protocol (15 minutes at 60°C followed by 30 minutes of rest with blankets). Control patients were provided with traditional therapy. At 60 months follow-up, 68.7% of the control group had suffered cardiac events due to heart failure or cardiac death, while significantly fewer (31.3%) sauna patients experienced an event (Kihara et al., 2009).

1.9 Heat shock proteins

Interestingly, heat shock proteins (HSPs) are biological molecules associated with cellular stress, and are in fact, preferentially increased after organisms are exposed to several stressors (Morimoto, 1998). More specifically, as their name implies, a primary stimulus for an increase in HSPs is elevated temperature or heat (Bathaie, Jafarnejad, Hosseinkhani, & Nakhjavani, 2010; Fujita et al., 2011). In fact, the preferential transcription of HSP genes was first discovered when *Drosophila* were exposed to high temperatures (Ritossa, 1962). An important characteristic of HSPs, the 70 kilodalton HSPs in particular, is that given their functions as molecular chaperones, assisting in protein folding, protein degradation and protein translocation (Beckmann, Mizzen, & Welch, 1990), cells and organs with elevated HSPs are better protected against future stresses. This is what is known as heat preconditioning, but could be used to describe any stress that fortifies an organism (e.g. exercise preconditioning).

While research has shown increases in Hsp70 to be beneficial, other studies suggest HSPs may be indicative of a diseased state, or even contribute to autoimmune disease (Alam et al., 2009; Nakhjavani et al., 2010). The examination of Hsc70 (the constitutive member of the HSP70 family) transgenic mice revealed that damage to pancreatic β -cells resulted in an increased incidence of Diabetes and a greater recruitment of serum cytokines and lymphocytes in Hsc70 transgenic mice (Alam et al., 2009). Examination of Hsp70 in diabetic humans shows significantly greater concentrations of serum Hsp70 in diabetics compared to non-diabetics (Nakhjavani et al., 2010), as well as higher levels of Hsp70 in patients who were diabetic for 5 years or longer (Nakhjavani et al., 2010). However, Gruden et al. (2009) and Milne and colleagues (unpublished

observations) have observed that very few individuals (<5%) express Hsp70 in the circulation, whereas the expression of antibodies against HSPs are in much greater concentration. Also, higher levels of anti-Hsp70 antibodies are correlated with improved odds ratios in Type 1 diabetics (Gruden et al., 2009). These mixed results regarding the potential benefits or detriments of increased Hsp70 reveal the need for further studies to determine the effects of these proteins.

There is also evidence to suggest that the induction of Hsp70 is sex-specific. After treadmill running, male rats have a twofold increase in cardiac Hsp70 compared to females (Paroo, Dipchand, & Noble, 2002). When this exercise was performed by ovariectomized females, increases in cardiac Hsp70 were similar to those in male rodents (Paroo, Haist, Karmazyn, & Noble, 2002). These differences extend to skeletal muscle as well. Post-exercise induction of Hsp70 at the protein and mRNA levels is significantly greater in male rats compared to females (Paroo, Dipchand, & Noble, 2002). Further, Shinohara et al. (2004) have observed that exogenous estrogen inhibits the heat induced accumulation of Hsp70 in rats. Since exercise may produce sex-specific increases in Hsp70 and post-ischemic cardiac function, it is of interest to determine whether heat stress is also associated with differences in Hsp70 induction between male and female humans.

Interestingly, the heat inducible 90 kilodalton HSP, Hsp90, has a role in endothelial nitric oxide synthase (eNOS) modulation (eNOS is an enzyme primarily responsible for the generation of NO in the endothelium) (García-Cardena et al., 1998; Gratton et al., 2000; Takahashi & Mendelsohn, 2003). This eNOS is maintained in an

inactive state by interaction with caveolin-1 protein (Navarro, Anand-Apte, & Parat, 2004) that upon stimulation is reduced, thereby freeing eNOS to bind to calmodulin (CaM) (Navarro et al., 2004). Takahashi & Mendelsohn (2003) suggest Hsp90 is involved in NO synthesis by interaction with Ca^{2+} /CaM. When eNOS is not saturated with CaM, Hsp90 decreases the EC_{50} (50% effective dose) for Ca^{2+} and CaM as well as dose dependently increasing the amount of CaM bound to eNOS (Takahashi & Mendelsohn 2003). Therefore, since eNOS is a CaM-dependent enzyme, Hsp90 increases NO synthesis and vasodilation. Also, it is possible that passive heating may improve vasodilation through a more direct Hsp90/eNOS mechanism since eNOS exists in a complex with Hsp90 in endothelial cells and the endothelial lining of intact blood vessels (Garcia-Cardena et al., 1998). It is suggested that shear stress acts as a stimulus to increase the molecular attraction of eNOS and/or Hsp90 for each other, increasing the association of proteins and the activation of eNOS (Garcia-Cardena et al., 1998). This activation may occur through the allosteric modulation of eNOS by Hsp90 via conformational change or stabilization of the eNOS dimer (Garcia-Cardena et al., 1998). Therefore, heat exposure may stimulate the Hsp90 activation of eNOS and improve vasodilation.

Further, HSPs play a role in exercise or heat preconditioning in mammals and it is thought that during exercise or heat stress, the accumulation of denatured proteins due to inherent cellular stress (e.g. increased temperature, reactive oxygen species, calcium transients, adrenergic signaling, etc.) increases the demand for chaperones (HSPs) and that an accumulation of HSPs in this regard produces cardioprotective effects (Noble, Milne, & Melling, 2008). For example, rats exposed to exercise training for as little as a

single 60 min bout that is concomitant with an increase in cardiac Hsp70 demonstrate improved post-ischemic cardiac function (Locke, Tanguay, Klabunde, & Ianuzzo, 1995). Paroo, Haist, Karmazyn & Noble (2002) also observed improved post-ischemic left ventricular developed pressure, maximal rate of contraction and relaxation, and a decrease in left ventricle end-diastolic pressure after treadmill running in rats. Similarly, rats exposed to longer training periods (e.g. daily running bouts over days to weeks) have improved post-ischemic recovery associated with increased myocardial Hsp70 (Locke et al., 1995; Le Page et al., 2009). However, the role of HSPs in physiological protection does not stop at the heart. In a study of streptozotocin-induced diabetic rats, hot tub therapy was associated with a significant increase in Hsp70 and decrease in advanced glycation end products (AGEs, which are markers of chronically elevated blood glucose, a negative consequence of diabetes), potentially due to Hsp70 reducing glycation (Bathaie, Jafarnejad, Hosseinkhani & Nakhjavani, 2010).

Since both exercise (see above) and heat stress (Bathaie et al., 2010; Fujita et al., 2011) stimulate the HSP response, either of these interventions may benefit the human population.

1.10 HSPs and diabetes

Diabetes is characterized by elevated blood glucose (hyperglycemia). Type I Diabetes (T1D) is an autoimmune condition, where the pancreas is not capable of producing the hormone insulin, which is responsible for glucose uptake in tissues. As a result, glucose accumulates in the blood, creating a hyperglycemic state. Type II Diabetes (T2D) occurs when the pancreas is incapable of producing sufficient amounts

of insulin, or various tissues are insensitive to insulin. Just as in T1D, glucose is unable to enter the cell and remains in the blood (Canadian Diabetes Association, www.diabetes.ca). In Windsor-Essex County, 7.2% of residents have been diagnosed with diabetes.

Interestingly, poorly managed diabetes increases an individual's risk of cardiovascular disease and approximately 80% of diabetics suffer a heart attack or stroke (Canadian Diabetes Association, 2011). The diabetic state is characterized by elevated triglycerides and decreased high density lipoproteins (HDL) (Dokken, 2008). Low density lipoproteins (LDL) are often smaller and more dense in diabetics, leading to a greater ability to infiltrate the arterial wall and become oxidized (Dokken, 2008). The oxidized particles attract immune cells, leukocytes, leading to atherosclerotic plaque formation (Chan, 1998). Furthermore, methylglyoxal (MG) is a glycation agent often present in greater quantities in diabetics (Rabbani et al., 2011). In vitro studies of MG-modified LDL (the equivalent that occurs in vivo) show decreased LDL particle size, increased proteoglycan binding and aggregation, indicating MG modification produces small, dense and more atherogenic LDLs (Rabbani et al., 2011).

Insulin is necessary for glucose uptake, and is important in regulating triglyceride (TRG) levels in the blood (Shen, 2007; Dokken, 2008). Insulin enhances the enzyme lipoprotein lipase, which cleaves triglycerides into free fatty acids (FFAs) for uptake into tissues (Shen, 2007). It also reduces hormone sensitive lipase, an enzyme that releases FFAs into circulation (Shen, 2007). With insufficient insulin production, as seen in diabetes, FFAs uptake is reduced and release into circulation is increased, resulting in

hypertriglyceridemia (Shen, 2007). This activity contributes to endothelial dysfunction, atherosclerosis and macrovascular disease.

Exercise and heat are both physiological stresses that enhance HSP production (Bathaie et al., 2010; Fujita et al., 2011; Lancaster & Febbraio, 2005; Ogawa et al., 2011; Walsh et al., 2003). Studies of diabetic animals show that Hsp70 is significantly lower in these groups when compared to healthy controls (Atalay et al., 2004; Yamagishi, Nakayama, Wakatsuki, & Hatayama, 2001). Furthermore, although exercise training increases Hsp70, diabetic animals show an attenuated response (Atalay et al., 2004). The same is true when Hsp70 is induced through hyperthermia as diabetic animals show impaired cytoprotective abilities (Yamagishi et al., 2001). However, an importance of timing of induction is indicated by the observation that Hsp70 protects against obesity-induced insulin resistance, such that mice exposed to heat therapy once per week over 16 weeks were protected against high fat diet induced hyperglycemia, hyperinsulinemia and elevated HOMA-IR (Chung et al., 2008). Diabetic humans also have altered HSP expression. At baseline, Kurucz et al. (2002) found a significant difference in Hsp-72 (Hsp70) mRNA concentration in muscle between diabetics and healthy controls, and a positive correlation between Hsp-72 mRNA and the rate of insulin stimulated glucose oxidation (Kurucz et al., 2002). Therefore, these results indicate that increasing Hsp70 may not only prevent the negatives of improper diet but also improve glucose handling.

Therefore, the benefits and potential use of circulating HSPs are not completely understood. While preliminary observations have been made on circulating Hsp70 (see

above), very little is known about Hsp90, even though it has an intimate relationship with the endothelium.

1.11 Sauna therapy and psychological well-being

Depressive and anxiety disorders are often treated with pharmacotherapy, however, exercise has been shown to be effective in the management of these disorders (Nahas & Sheikh, 2011; Helmich et al, 2010; Babyak et al., 2000; Broman-Fulks et al., 2004; and Broocks et al., 1998). For example, a significant reduction in Hamilton Rating Scale for Depression scores suggests the role of exercise as a monotherapy or adjunct to traditional treatments for depression (Nahas & Sheikh, 2011). Furthermore, moderate intensity exercise 3 times per week is effective in the treatment of patients with major depression (Babyak et al., 2000). These benefits extend to anxiety disorders as well, as Broman-Fulks et al. (2004) show that high and low intensity exercise significantly reduced anxiety sensitivity. In addition, regular aerobic exercise is associated with significant clinical improvements in patients with panic disorder when compared to placebo (Broocks et al., 1998). The proposed mechanisms underlying these improvements include increasing serotonin and central norepinephrine neurotransmission as well as raising the concentration of endorphins (Helmich et al., 2010). Interestingly, endorphins are involved in heat adaptation in the central nervous system (Vescovi & Coiro, 1993) and have been shown to attenuate the physiological response to thermal stimuli (Holaday, Wei, Loh & Li, 1978). Based on the role of endorphins in both exercise and heat stress, it is possible that thermal therapy may produce some of the similar psychological benefits as exercise. TRPV3 thermosensitive receptors, when selectively activated have anxiolytic and antidepressant effects in mice (Moussaieff et al.,

2008). Whether passive heating has the same effect is unknown, however an uncontrolled study of bamboo charcoal kin use in Japan revealed lower anxiety levels among participants following heat exposure (Hayasaka et al., 2008). Others have measured salivary cortisol, a hormone elevated during physical and emotional stress, before and after spa bathing with 42°C water (Toda, Morimoto, Nagasawa, & Kitamura, 2006). Cortisol was lower following bathing, with a more pronounced effect in those with higher levels prior to bathing (Toda et al., 2006).

Masuda et al (2005) examined the effects of daily infrared sauna treatment in depressed inpatients. Results showed a reduction in somatic complaints, and improvements in hunger and relaxation scores (Masuda et al., 2005). In addition, significant increases in plasma ghrelin (a hunger hormone) concentration and daily caloric intake were observed (Masuda et al., 2005). Research on the effects of passive heating in depressive and anxiety disorders is limited, and further studies are required to better understand these effects.

1.12 Risks of heat therapy

While outcomes show the potential benefits of sauna therapy, risks are often cited (Kukkonen-Harjula & Kauppinen, 2006; Press, 1991). Consequently, it is essential to ensure this treatment is safe. Basford et al. (2009) examined the effects of sauna bathing in New York Heart Association class III and IV CHF patients. Participants underwent sauna bathing three times a week over four weeks in a 60°C sauna for 15 minutes each day. The intervention was found to be safe and well-tolerated in this group (Basford et al., 2009). Furthermore, sudden deaths resulting from sauna baths are infrequent,

especially in younger populations. Among men 60 years and older, sauna related deaths occurred at a rate of 1 in 0.4 million baths (Kukkonen-Harjula & Kauppinen, 2006).

These incidents are less frequent in the 50-59 year group (1 in 2.3 million baths), and are least likely to occur among 40-49 year olds (1 in 9 million baths) (Kukkonen-Harjula & Kauppinen, 2006).

1.13 Summary

In conclusion, the effects of exercise on markers of health and disease prevention are well known. However, only half of Windsor-Essex county residents meet the recommended daily physical activity guidelines. Sauna therapy may be an effective method for reducing the negative health outcomes associated with physical inactivity, for the reasons described above but also because improvements in vascular function have been observed after only 75 minutes of sauna exposure per week (Kihara et al., 2002, 2009; Miyata & Tei, 2010). This is half of that recommended if using exercise (~150 minutes). Further, cardiovascular improvements such as flow-mediated dilation (FMD) (Kihara et al., 2002; Nguyen, Naseer, & Frishman, 2004), and improved endothelial nitric oxide synthase (eNOS) production (Ikeda et al., 2001; Ikeda et al., 2005) have been reported after as little as only 2 weeks of thermal therapy, and heat therapy may even be beneficial for individuals with metabolic diseases such as Diabetes. Improvements in glycemic control such as enhanced insulin sensitivity and improved fasting plasma glucose have been observed after a similarly short duration of hot tub therapy (Hooper, 1999). As such, it is essential to investigate alternative approaches to exercise and pharmacotherapy in an attempt to reduce the risk of cardiovascular and metabolic disease among adults, especially those more at risk for developing chronic diseases but before the

spikes in chronic disease have occurred (i.e. middle-aged). Thermal therapy in the form of daily sauna bathing may provide an alternate or adjunct prevention strategy against these risk factors.

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Chapter 2

2.1 Introduction

Cardiovascular disease (CVD) is the second leading cause of death among Canadians (Statistics Canada, 2011), with heart disease and stroke accounting for 21.3% and 5.8%, respectively, of all deaths in 2008 (Statistics Canada, 2011). These adverse cardiovascular events cost the Canadian economy an estimated 20.9 billion dollars in 2008 (Heart and Stroke Foundation, 2011), placing an enormous burden on health care systems. Fortunately, CVD has a number of modifiable risk factors including hypertension, blood cholesterol, diabetes, obesity, physical inactivity, smoking and stress (Heart and Stroke Foundation, 2011). Lifestyle modifications such as diet and exercise effectively reduce the risk of developing CVD (Chobanian et al., 2003). Despite these benefits, many Canadians fail to meet the recommended 150 minutes of moderate to vigorous exercise each week (Canadian Society for Exercise Physiology, 2012; Statistics Canada, 2011), and many of those already diagnosed with risk factors such as hypertension fail to exercise regularly (Uzun et al., 2009). Furthermore, musculoskeletal injuries and fear of becoming injured also limit exercise participation (Perri et al, 2002). Interestingly, when lifestyle modifications are not sufficient to reduce blood pressure, cholesterol or blood glucose, pharmacotherapy may be introduced, but still, adherence remains problematic. For example, antihypertensive drug use adherence is influenced by age, gender and cost among other factors (Munger et al., 2007).

Therefore, sauna bathing may be a suitable alternative for individuals who are unable to participate in regular physical activity. Sauna treatment in clinical populations and individuals with coronary risk factors has been associated with symptom

improvement and reduced risk of cardiovascular disease (Kihara, 2004; Kihara, 2009; Imamura, 2001). Chronic heart failure (CHF) patients who underwent 2 weeks of daily 15 minute sauna treatment showed improved HRV (increased SDNN), and fewer premature ventricular contractions (Kihara, 2004). In an investigation of the long-term effects of sauna bathing among CHF patients, Kihara et al. (2009) reported fewer cardiac events after 60 months. Vascular endothelial function has also been shown to improve following sauna treatment, as percent flow mediated dilation (%FMD) among individuals with 1 or more coronary risk factor significantly improved (Imamura et al., 2001). Furthermore, improvements in glycemic control such as enhanced insulin sensitivity and improved fasting plasma glucose have been observed after short duration of hot tub therapy (Hooper, 1999), and sauna bathing may improve measures of psychological health. For example, spa bathing with 42°C water has been associated with lower cortisol (a stress hormone) levels (Toda, Morimoto, Nagasawa, & Kitamura, 2006) and the effects of thermal therapy on depressed patients are also promising since daily infrared sauna treatment of depressed patients results in fewer somatic complaints, and improved hunger and relaxation scores (Masuda et al., 2005).

The mechanisms underlying these improvements are still unconfirmed. Given the physiological response to passive heating (i.e. elevated heart rate and cardiac output, increased sweating and ventilation) it is possible that these improvements occur in the same manner as with aerobic exercise. In addition, it is unknown whether the benefits of sauna bathing extend to a generally healthy population, indicating a need for further study. Should sauna exposure improve CVD risk factors, it may serve as an alternative or adjunct to traditional lifestyle interventions aimed at reducing the onset of CVD.

Objectives

The primary objective of this experiment is to determine whether short-term sauna treatment improves markers of cardiovascular, metabolic and psychological health in sedentary middle-aged males and females.

2.2 Hypothesis

We hypothesize that passive heating will be associated with health improvements as it is similar to exercise stress. Changes in blood flow, ventilation, and sweat production occur during acute exercise as well as passive heating. As such, we predict the following:

- 1) Two weeks of daily sauna treatment will improve markers of metabolic, cardiovascular and psychological health.

2.3 Methods

Participants

To examine the physiological and psychological adaptations that occurred with 2 weeks of sauna use, middle-aged males (n=2) and females (n=3) were recruited from the Windsor-Essex Region including the University of Windsor campus. Participant characteristics are presented in Table 2.1.

Table 2.1. Participant characteristics.

Measure	Mean	±	SD
Age (y)	46.4	±	9.07
Female (n)		3	
Male (n)		2	
Height (cm)	169.2	±	0.42
Weight (kg)	73.84	±	10.86
Body Mass Index (kg/m ²)	25.66	±	1.21
Body Fat %	28.56	±	9.56
Fat Mass	25.93	±	14.42
VO ₂ peak (ml/kg/min)	20.76	±	7.42

Informed Consent and Initial Questionnaires

Prior to testing, all participants were seated with an informed consent document. They were provided a verbal explanation of the purpose, risks, benefits, and protocol and were asked to read the consent form prior to signing. Participants were also informed that they were free to withdraw from the study at any point with no consequences. Testing occurred in the Physical Activity and Cardiovascular Research (PACR) laboratory at the University of Windsor. Upon entering the lab, participants were seated and the experiment and risks were explained. Participants were then required to read and sign informed consent, complete a Physical Activity Readiness Questionnaire (PAR-Q, Appendix A), complete a Participant Information Sheet (Appendix B), and complete a Risk Factor and Medication sheet (Appendix C) before participation in the study. Participants were excluded if they met any of the criteria outlined by Nguyen et al., 2004 (Table 2.2), or if they had any limiting medical conditions or were taking medications that may have caused negative effects during sauna bathing. All participants were asked not to make any lifestyle changes (ie. diet, exercise) for the duration of the study and follow-up period. This protocol was approved by the Research Ethics Board at the University of Windsor.

Table 2.2. Exclusion criteria to sauna use.

Exclusion criteria		Rationale
Limiting physical conditions	<ul style="list-style-type: none"> • Alcohol consumption • Severe aortic stenosis, unstable angina pectoris, recent myocardial infarction, decompensated heart failure, and cardiac arrhythmias. • Hypotension in patients on β-blocker and short-acting nitroglycerin • Orthopedic conditions that prevent the completion of a cycle ergometer test. • 2 or more cardiovascular disease risk factors. • Pregnancy. 	<ul style="list-style-type: none"> • Increases the risk of hypotension, fainting, and arrhythmias (Roine et al, 1992). • Increase the risk of hypotension, fainting, re-infarction, arrhythmias, and sudden death (Keast et al, 1988; Eisalo et al, 1988; Luurilia, 1992) • Iisalo et al, 1969; Luurila et al, 1989; Barkve et al, 1986
Medications	<ul style="list-style-type: none"> • Diuretics • Anticholinergics • Barbiturates • Beta-blockers • Antihistamines 	<ul style="list-style-type: none"> • All may impair heat loss and increase the risk of heat related illness.

Criteria were established from those outlined by Nguyen et al. 2004.

Design

Following initial screening, participants underwent baseline blood pressure and HRV measurements as part of the familiarization process. Participants were then asked to return to the lab within 1 week for baseline physiological and psychological testing (resting HR, BP, blood glucose, hematocrit, State Trait Anxiety Inventory (STAI) and VO_2). These physiological and psychological tests were completed on 3 separate occasions: baseline, after 2 weeks of normal activity and immediately prior to sauna therapy and 2-3 days after 2 weeks of sauna therapy. In this regard, participants acted as their own controls for the initial 2 week baseline period. A flowchart of the study timeline is diagrammed in Figure 2.1. On each testing day, participants were asked to report to the PACR lab after an 8 hour fast. Once there, the following procedures were completed in the order of their appearance.

Figure 2.1

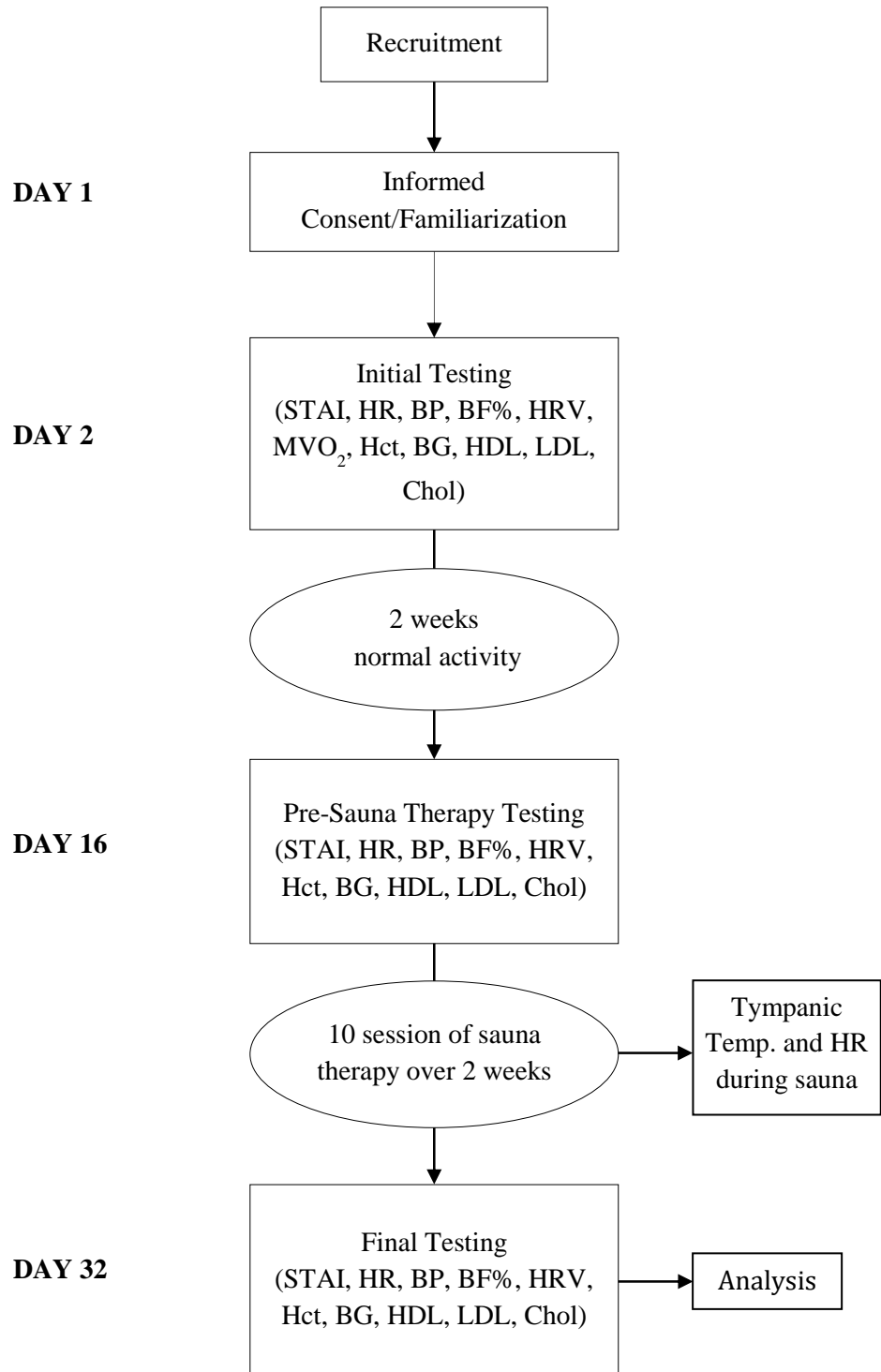


Figure 2.1. Flow diagram of study procedures. Note that peak VO₂ analysis was only performed on the first testing session. STAI (State Trait Anxiety Inventory), HR (resting heart rate), BP (resting blood pressure), BF% (percent body fat), HRV (heart rate variability), Hct (hematocrit), BG (blood glucose), HDL (high density lipoprotein), LDL (low density lipoprotein), Chol (cholesterol).

Testing Days

State Trait Anxiety Inventory (STAI)

After the initial meeting and subsequent to informed consent, Group II participants were seated and required to complete the STAI (Appendix C). They were instructed to follow the instructions on the form and the researcher calculated the scores at a later time. The STAI is designed to measure temporary state anxiety and more general and long-standing trait anxiety. The inventory consists of 2 sections, 20 questions each using 4 point Likert items. The number on the Likert scale is positively correlated with the anxiety related to the question. The STAI validity has been assessed, and is correlated with the Anxiety Scale Questionnaire and Manifest Anxiety Scale with correlation scores of .73 and .85, respectively (Spielberger, Reheiser, Ritterband, Sydema & Unger, 1995). Furthermore, Rule and Traver (1983) determined the reliability of the State Anxiety measure to be .40 and Trait Anxiety measure to be .86.

Heart Rate Variability (HRV)

Participants reported to the lab following an 8 hour fast as well as having refrained from caffeine consumption for 8 hours. The investigator placed 3 electrodes on the participant's chest, one below each clavicle and one on the lower left side of the abdomen. The participant lay supine on a bed for 20 minutes in a quiet temperature controlled room with the lights dimmed. Following the rest period, 10 minutes of electrocardiograph data was recorded for later analysis. R-R interval data was recorded at a sampling frequency of 1000 Hz, and signals were converted to digital using a data acquisition board (PowerLab 8/30, ADInstruments Inc., Colorado Springs, CO, USA).

Recordings were edited using an ECG editor program to detect R-R intervals. R-R interval data was then analyzed using an HRV analysis program.

Blood pressure, body composition

Resting arterial blood pressure (BP) and resting heart rate (HR) were assessed after 10 minutes of seated rest using a non-invasive automatic device (DinamapProcare 200/Carescape 200, GE Healthcare Canada, ON, Canada). 4 measurements were obtained, the first was discarded and the remaining 3 were averaged.

Body composition was measured using air-displacement plethysmography (BodPod, Life Measurements Inc., Concord, CA, USA). Prior to entering the BodPod, participant height was recorded. Participants were instructed on the breathing protocol, asked to remove excess clothing and jewelry and wear the appropriate cap. Females wore either a swimsuit or spandex shorts and bra and males wore athletic shorts. Once the participants received instructions and weight was measured, they entered the BodPod and followed on-screen breathing instructions.

Fasting plasma glucose (FPG), blood lipids

Blood glucose, total cholesterol, high density lipoproteins (HDL), triglycerides, low density lipoproteins (LDL), non-HDL, and LDL/HDL were measured using a Cholestech LDX system (Cholestech LDX®, Cholestech, USA) and fingerstick blood. For each sample, the investigator wore latex gloves, inserted a new needle into the lancing device (Accu-Check Softclix, Roche Diagnostics) and swabbed the participant's skin with an alcohol swab. The blood drop was collected in an anticoagulant coated capillary tube (Cholestech) and a plunger was inserted to force the blood into the

collection tray for analysis. This test is highly comparable to The Centers for Disease Control and Prevention's Cholesterol Reference Method and other laboratory tests.

Hematocrit

A second set of capillary tubes were used to collect fingerstick blood .

Hematocrit was measured using a microhematocrit centrifuge (Statspin CritSpin, Iris Sample Processing, USA) with manual card reader.

Maximal oxygen consumption (VO_2peak)

Participants were fitted with a telemetric heart rate monitor (Polar, Canada).

Participants were asked to mount an Ergoline cycle ergometer and seat height and handle bar distance were adjusted. A mouthpiece for gas analysis was fitted with a head strap.

The subject was asked to begin pedaling at 80 rpm and to maintain this pace for the entire test. If the participant fell below 60 rpm, the test was terminated. Volume of oxygen consumed (VO_2), volume of carbon dioxide consumed (VCO_2), respiratory exchange ration (RER) and heart rate (HR) were recorded throughout the test using an online gas analysis system (MediSoft, Ergocard). After 2 minutes of unloaded cycling, the resistance was increased in an incremental fashion 25W/min until volitional exhaustion.

Throughout the test, verbal encouragement was given in an attempt to help the participant achieve maximal effort. Maximal effort was determined if 2 of the following 4 criteria were met: a heart rate within 10 beats of age predicted maximum; inability to maintain 60 rpm on the cycle ergometer; a plateau in VO_2 even though resistance had been increased; a respiratory exchange ratio greater than or equal to 1.1. Moreover, the test was stopped if the participant exhibited any physical signs of distress (such as pale skin, confusion, a reduction in heart rate with an increase in intensity, pains in the chest, etc.) that would

have been indicative of an inability to meet the demands of exercise. After completion of the test, the subject was required to remain on the cycle, with no resistance until heart rate returned to within 10 beats of the initial stage. Subsequently, the mouthpiece was removed and the subject was asked to dismount the cycle. The $\text{VO}_{2\text{peak}}$ test was performed only on the first testing day. The remaining 3 testing days included all physiological measures except $\text{VO}_{2\text{peak}}$.

Sauna treatment

Female participants reported to the Windsor Squash and Fitness Club, and male participants to the University of Windsor St. Denis Centre for 10 visits over 14 consecutive days, and were permitted to miss only one session if necessary. Prior to entering the sauna, tympanic temperature and weight were measured. Participants wore either a swimsuit, or spandex shorts and bra and were fitted with a telemetric heart rate monitor and watch to record heart rate throughout the sauna bath (Polar, USA). The sauna thermostat was set to approximately 80-90°C to account for other fitness center members entering and leaving the sauna throughout the participant's session. Actual sauna temperature was recorded between 50-60°C during all sauna baths.

Participants were informed to remain seated comfortably in the sauna and reminded that they were free to exit the sauna at any time should they experience any discomfort (ie. dizziness), and bottled water was available throughout the testing session. Tympanic temperature was recorded before, midway through, and upon completion of the 15 minute sauna bath (Braun ThermoScan 5 IRT4520). Upon exiting the sauna bath, the participant was weighed to account for sweat loss. The participant was asked to lie on a massage table while covered with a thermal blanket (SOL thermal bivvy) for 30 minutes.

Tympanic temperature was recorded every 5 minutes during this period. Central thermoreceptors located in the brain detect changes in blood temperature. Therefore tympanic measurement was used as it provides an estimate of the temperature of blood entering specific brain centres (Lee et al., 2011). Participants were permitted to leave when heart rate was within 10 beats of the initial stage. Sauna treatment was repeated for 10 days over a 2 week period as has been previously outlined. Within 2-3 days of the final sauna treatment, participants returned to the PACR lab for post-sauna testing which included the same measures outlined above except for VO_2 testing.

Statistics

Statistical analysis was performed using PASW version 18.0 (or latest version). All data including descriptive statistics are presented as means \pm standard deviations (SD). A repeated measures ANOVA (RM ANOVA) was performed to compare baseline, pre-treatment, and post-treatment measures. Upon finding significant main effects, individual group means were compared using Least Significant Difference (LSD) post hoc analysis. In a recent study, Kihara et al. (2002) examined the effects of sauna treatment for men and women with CHF and did not note any sex differences in the ability of the therapy to improve cardiovascular health. Initial fitness (as measured by $\text{VO}_{2\text{peak}}$) and body fat percentage were analyzed as covariates. Mean differences were considered statistically significant where $p < 0.05$.

2.4 Results

Physiological Responses to Passive Heating

Tympanic temperature and heart rate were recorded for each sauna session. Complete data was available for only 4 participants. Tympanic temperature increased significantly among all participants (mean baseline temperature $36.0^{\circ}\text{C} \pm 0.8$ vs mean post-sauna temperature $37.9^{\circ}\text{C} \pm 0.6$, $p < 0.001$; Table 2.3). This change in tympanic temperature occurred during the time in the sauna, but was maintained at approximately 1°C higher than pre-sauna values for the 30 minute period under the thermal blanket (Table 2.3, Figure 2.2).

Heart rate was significantly increased by approximately 10 beats per minute during each of the 10 sauna exposures, but quickly returned to pre sauna values after exiting the sauna and remained this way for the 30 minute period under the thermal blanket (Figure 2.3).

Markers of Health

Significant differences were observed for systolic and diastolic blood pressure (systolic baseline 107.4 ± 13.9 mmHg, pre-sauna 99.2 ± 15.7 mmHg, and post-sauna 100 ± 11.8 mmHg, $p < .05$); diastolic baseline 65.6 ± 5.8 mmHg, pre-sauna 60 ± 7.9 mmHg, and post-sauna 60.8 ± 8.2 mmHg, $p < 0.05$), (Table 2.4). No significant changes were observed for resting heart rate, body mass, body fat percentage, total cholesterol, high density lipoproteins, triglycerides, low density lipoproteins, non-high density lipoproteins, or blood glucose (Table 2.4). Hematocrit was significantly increased following 10 days of sauna treatment (baseline 41.0 ± 5.0 , pre-sauna 41.6 ± 4.6 , and post-sauna 43.0 ± 4.6 , $p = 0.006$) (Table 2.4).

Table 2.3. Mean sessional tympanic temperatures.

Participant	Tympanic Temperature (°C)					
	Pre-Sauna		Post Sauna		Post Session	
1	36.6	± 0.4	38.1	± 0.5	37.1	± 0.1
2	35.7	± 0.5	37.8	± 0.4	36.8	± 0.3
3	36.5	± 0.3	38.5	± 0.4	36.8	± 0.4
4	35.0	± 0.5	37.0	± 0.5	36.4	± 0.2
5	37.1	± 0.3	38.3	± 0.6	37.4	± 0.3
Mean	36.0	± 0.8	37.9	± 0.6*	36.9	± 0.4

Average 10 day tympanic temperatures for each participant (n=5). Pre-sauna= prior to entering sauna, post-sauna= upon exiting sauna, post session= following 30 minute covered rest. (* p<0.001)

Figure 2.2

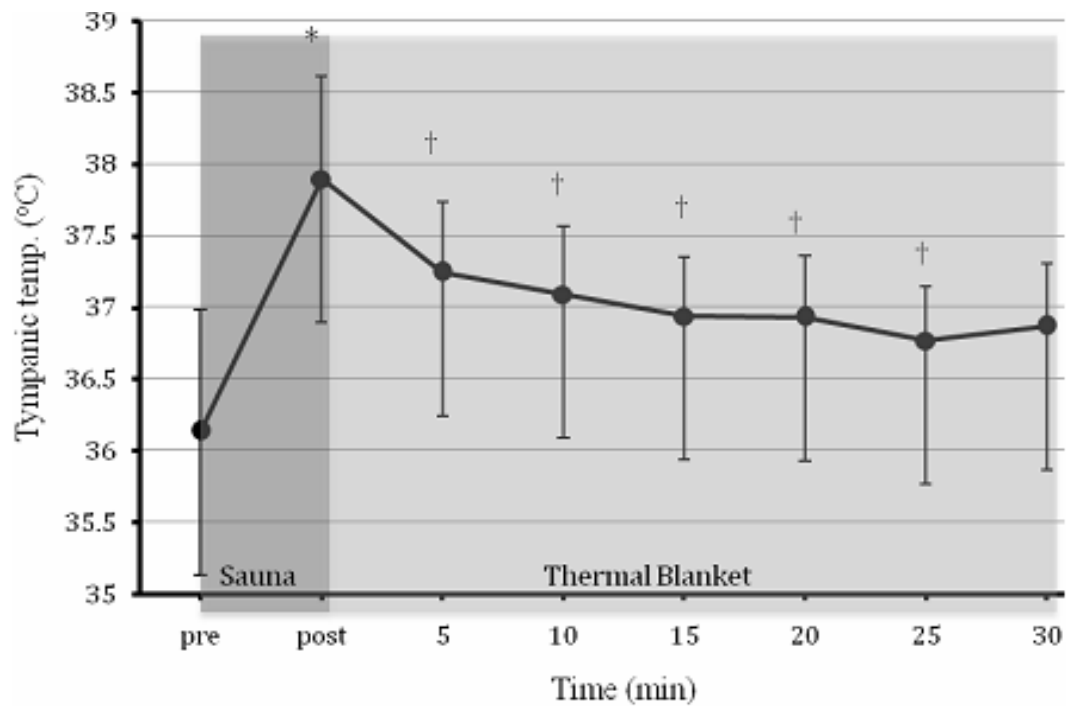


Figure 2.2. Tympanic temperature during sauna exposure and rest (n=5). * $p < 0.001$ between pre- and post-sauna temperature. Each data point represents the mean \pm standard deviation. † = $p < 0.05$ compared to pre-sauna temperature. $p = 0.051$ at 30 minutes compared to pre-sauna.

Figure 2.3

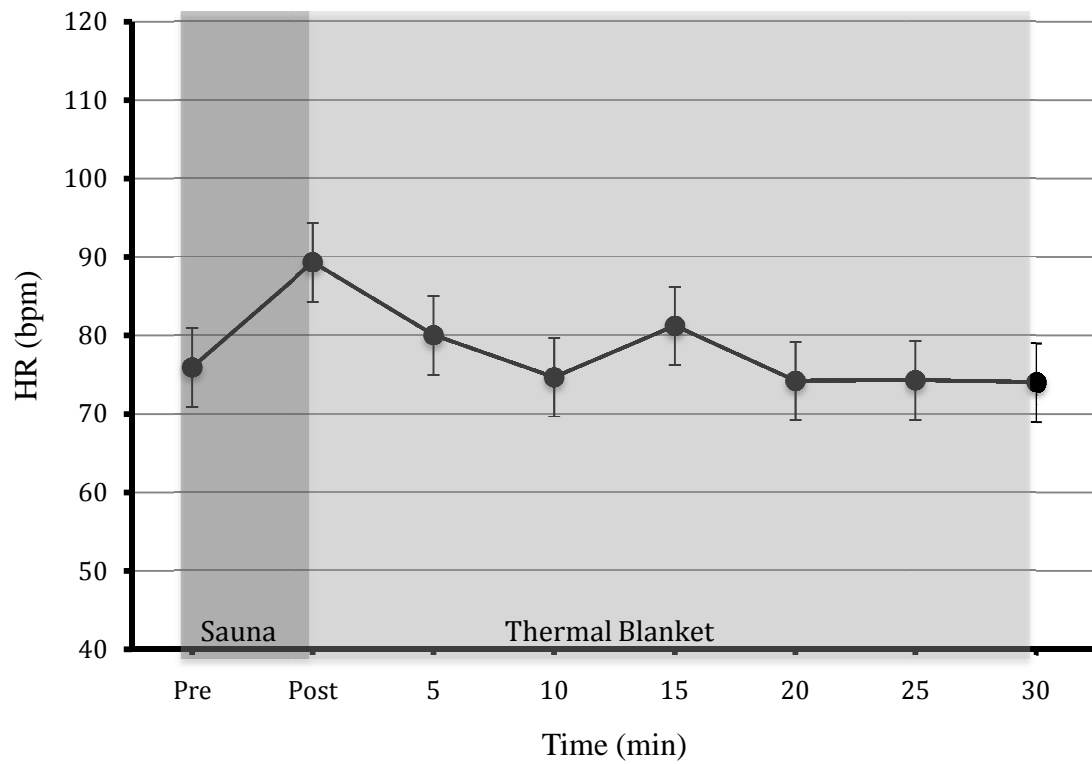


Figure 2.3. Average 10-day heart rate response at rest, immediately following sauna bath, and during covered rest (n=5). Each data point represents mean \pm standard deviation. No significant differences ($p=0.056$).

Table 2.4. Physiological measures of participants at baseline, prior to sauna treatment (pre-sauna), and following 2 weeks of sauna treatment (n=5).

Measure	Baseline	Pre-Sauna	Post-Sauna	p
SBP (mmHg)	107.4 ± 13.9	99.2 ± 15.7 †	100.0 ± 11.8	0.028
DBP (mmHg)	65.6 ± 5.8	60.0 ± 7.9 †	60.8 ± 8.2	0.027
Resting HR	67.6 ± 7.0	65.6 ± 4.0	62.4 ± 4.6	0.404
Body mass (kg)	73.8 ± 10.9	74.5 ± 10.5	74.3 ± 10.8	0.627
Body fat (%)	28.6 ± 9.6	29.4 ± 9.6	29.3 ± 9.9	0.732
Total Cholesterol (mg/dL)	185.6 ± 81.3	228.0 ± 49.9	213.0 ± 49.1	0.480
HDL (mmHg)	68.2 ± 17.5	67.0 ± 22.6	62.8 ± 11.1	0.903
Triglycerides (mg/dL)	118 ± 70.4	109.0 ± 37.4	100.6 ± 29.8	0.740
LDL (mmHg)	133.8 ± 35.6	136.5 ± 49.0	130.0 ± 46.5	0.877
non-HDL (mg/dL)	157.2 ± 47.9	160.3 ± 54.5	150.0 ± 51.9	0.820
Glucose (mg/dL)	83.4 ± 7.4	82.2 ± 11.0	81.4 ± 9.9	0.903
Hematocrit (%)	41.0 ± 5.0	41.6 ± 1.91	43.0 ± 4.6*	0.006

Data are presented as means and standard deviations. *=significantly different from baseline and pre-sauna therapy, p<0.05. †= significantly different from baseline, p<0.05. SBP (systolic blood pressure), DBP (diastolic blood pressure), HDL (high density lipoprotein), LDL (low density lipoprotein), non-HDL (non-high density lipoprotein).

Reference values (mg/dL):

Total cholesterol: desirable (<200), borderline high (200-239), high (≥240)

HDL: low (<40), high (≥60)

LDL: optimal (<100), borderline high (130-159), high (160-189)

Triglycerides: normal (<150), borderline high (150-199), high (200-499)

Fasting plasma glucose: normal (<100)

Heart Rate Variability

Heart rate variability (HRV) data was not available for only one male participant due to an unforeseen technical malfunction. Nonetheless, there were no significant differences observed for any component of HRV (Table 2.5).

Psychological Measures

The State-Trait Anxiety Inventory (STAI) was completed at baseline, 2 weeks later, and upon completion of 2 weeks of sauna exposure. There were no significant differences for state or trait anxiety scores across trials (Table 2.6).

Table 2.5. Heart rate variability measures for participants (n=5), before sauna treatment (pre-sauna) and following 2 weeks of sauna treatment (post-sauna).

Measure	sex ID	Participant				Mean	±	SD	p
		f 1	f 2	f 3	m 4				
Mean	pre-sauna	1003	963.3	889.8	942.3	949.6	±	47.1	0.721
	post-sauna	935.6	927.2	1062	757	920.5	±	125.2	
SD	pre-sauna	73.62	127.2	58.71	46.58	76.53	±	35.55	0.429
	post-sauna	67.83	61.72	80.3	29.88	59.93	±	21.48	
VLF	pre-sauna	1142.79	574.8	712.81	349.85	695.06	±	333.87	0.526
	post-sauna	1136.35	729.69	1098.09	163.9	782.01	±	451.02	
LF	pre-sauna	1409.94	740.94	427.08	429.72	751.92	±	462.76	0.619
	post-sauna	1015.19	791.59	1842.61	259.34	977.18	±	658.32	
HF	pre-sauna	294.39	127.34	484.26	95.61	250.4	±	178.63	0.379
	post-sauna	324.09	81	480.18	23.34	227.15	±	213.17	
Total Power	pre-sauna	3264.6	1443.09	1624.15	875.18	1801.76	±	1026.1	0.422
	post-sauna	2475.63	3755.63	3420.88	446.57	2524.68	±	1487.66	
LF:HF	pre-sauna	4.79	5.82	0.88	4.49	4	±	2.15	0.184
	post-sauna	3.13	9.77	3.84	11.11	6.96	±	4.06	
HF:TP	pre-sauna	0.1	0.09	0.3	0.11	0.15	±	0.1	0.193
	post-sauna	0.13	0.02	0.14	0.05	0.09	±	0.06	

Data are presented as means for each participant. Data are unavailable for 1 male participant due to unforeseen technical errors. Means ± standard deviations are presented for the group. There were no significant differences in any of the HRV measures. P-values represent pre versus post for each measure. SD (standard deviation of NN intervals), VLF (very low frequency), LF (low frequency), HF (high frequency), LF:HF (low frequency to high frequency ratio), HF:TP (high frequency to total power ratio).

Normal values (Mahk, 1991):

Total power (ms²): 3466 ± 1018

Low frequency power (ms²): 1170 ± 416

High frequency power (ms²): 975 ± 206

Table 2.6. State and trait anxiety measures for participants at baseline, prior to sauna treatment (pre-sauna), and following two weeks of sauna treatment (post-sauna). (n=5).

Measure	Baseline	Pre-Sauna	Post-Sauna	p
State	49.8 \pm 4.60	49.6 \pm 2.30	49.8 \pm 1.79	0.994
Trait	47.4 \pm 3.58	46.2 \pm 1.64	45.6 \pm 2.30	0.396

Data are presented as means \pm standard deviations. There were no significant differences between any time points.

Acute Vs. Chronic Effects of Sauna Bathing

In order to determine whether there were differences between a young healthy population and repeated exposures of a middle-aged population, a separate group of participants was recruited and subjected to only 1 sauna session as described in the methods. 5 male subjects (aged 19.89 ± 2.1 years) agreed to participate before closure of the St. Denis Centre sauna. The heart rate response to passive heating did not differ between the young or middle aged groups (Figure 2.4), however the middle-aged group showed greater tympanic temperature elevations ($p < 0.05$; Figure 2.5).

Figure 2.4

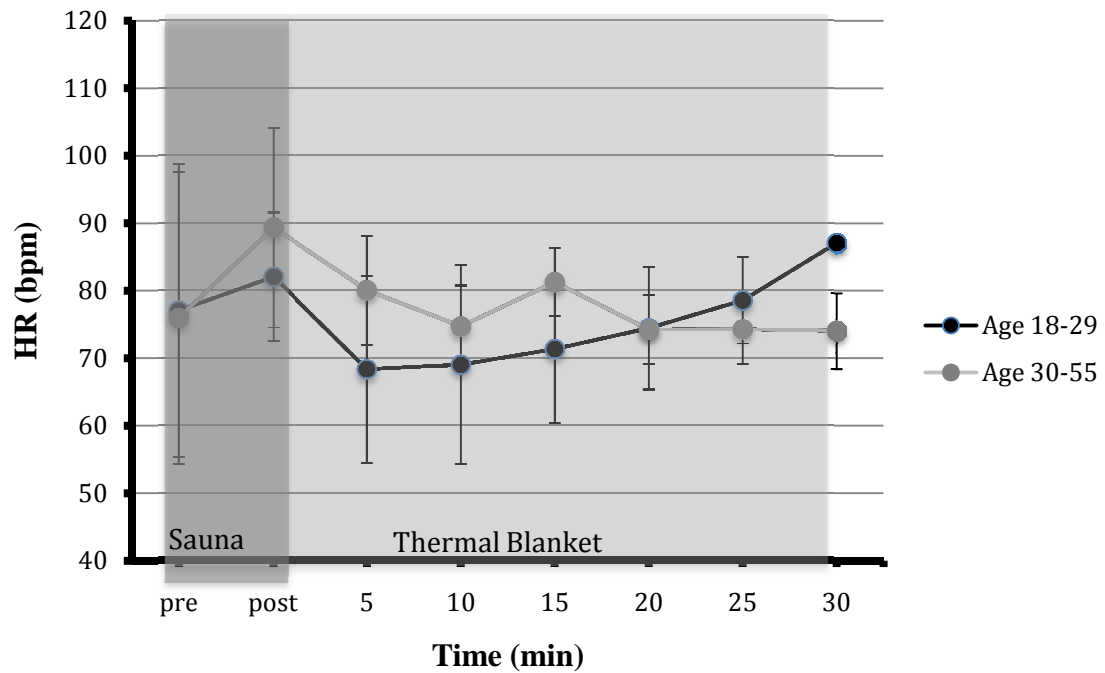


Figure 2.4. Average heart rate during a single sauna bath and covered rest among 18-29 year old participants (n=5), and the 10 day average heart rate during sauna bath and covered rest among 30-55 year old participants (n=5). Each data point represents the mean \pm standard deviation. There were no significant differences between groups ($p=0.515$).

Figure 2.5

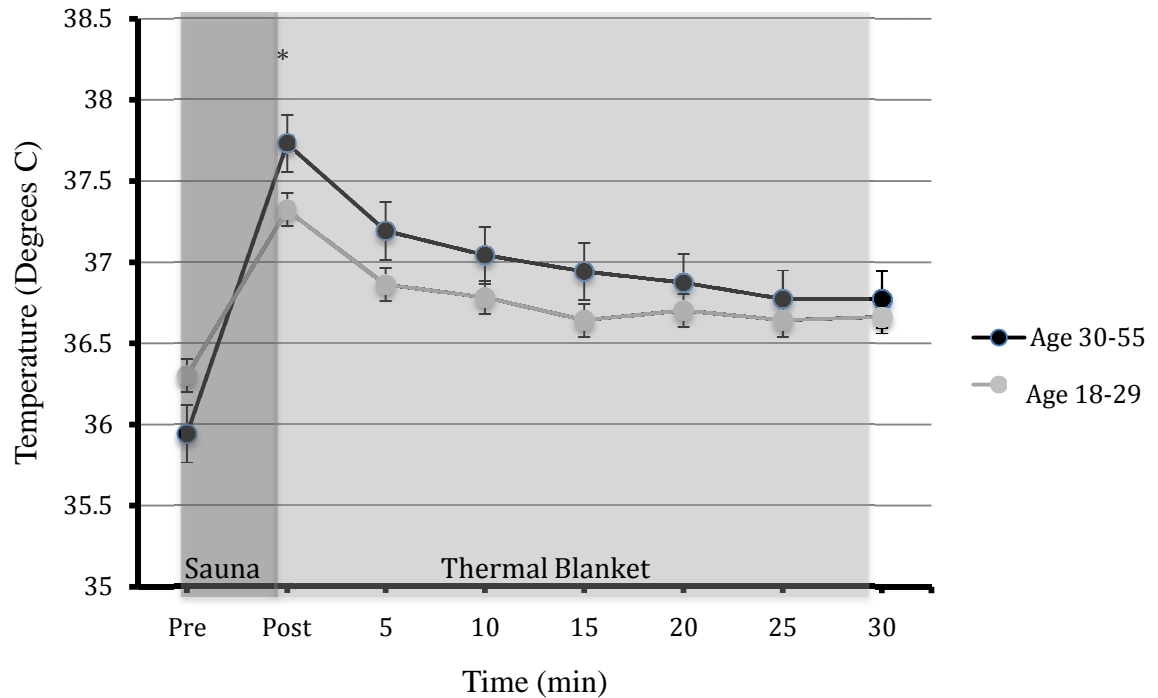


Figure 2.5. Average tympanic temperature during a single sauna bath and covered rest among 18-29 year old participants (n=5), and the 10 day average tympanic temperature during sauna bath and covered rest among 30-55 year old participants (n=5). Each data point represents the mean \pm standard deviation. There was a significant main effect for age such that the 30-55 year old participants exhibited significantly higher tympanic temperature in response to sauna exposure ($p<0.05$).

2.5 Discussion/Conclusions

Since increasing age is associated with the development of cardiovascular risk factors and an increased risk of adverse events and CVD, we utilized a sauna bathing intervention similar to that of Kihara et al (2004) to determine whether sauna treatment could improve various health markers. Each sauna session elevated core temperature by approximately 1.5°C and then maintained an approximately 1°C increase above pre-sauna values throughout the 15 minute sauna period and the 30 minute covered rest. Further, HR was elevated by approximately 10 bpm immediately following each sauna bout. As such, we predicted training effects following a 2 week intervention, however, heart rate variability (HRV) and other health markers did not show improvements following 2 weeks of sauna use. Subsequent to secondary analysis, we noted that middle-aged participants attained greater tympanic temperatures compared to young participants, which would be indicative of a greater heat stress. Nonetheless, factors such as sample size, and the degree of heat stress may have accounted for the lack of significant improvements in these health markers.

The primary measure of this study was heart rate variability. We anticipated improvements in HRV following 2 weeks of sauna use, however we observed no significant differences between pre- and post- treatment measures. The small sample size may account for the lack of statistical significance, and may be of particular importance with respect to female participants as HRV is influenced by menstrual cycle phase (Sato et al, 1995). Specifically, HF power tends to decrease during the luteal phase, while the LF:HF ratio increases. We attempted to account for these changes by documenting the approximate last menstrual cycle start date at the onset of the study. Based on the self-

report, we determined testing occurred during the follicular phase for one participant, the luteal phase for another, and was unknown for the third, as she experienced amenorrhea within the recent months leading up to the study. Despite taking this information into account, our results did not follow the trends proposed by Sato et al (1995). It is possible that participants provided inaccurate reports of menstrual cycle start date. However, the male participant and the amenorrheic female participant also showed no improvement in the HRV measures, suggesting menstrual cycle phase may not be the sole factor influencing HRV.

The time of measurement may also account for the differences between our results and those reported by Kihara et al (2004). Participants underwent final physiological and psychological testing, including ECG recording between 2 and 3 days following the final sauna treatment. Kihara et al (2004) performed ECG recording the day after the final treatment. Consequently, it is possible that the ECG improvements are transient and that the brief period (2 weeks) of sauna exposure is not able to provide lasting adaptations. Moreover, another possible difference between the data reported by Kihara et al (2004) and those of the present study may have been in the length of time of HRV collection. In the present study, a short-term measure of HRV was performed under standard laboratory conditions. Kihara et al (2004) analyzed a minimum of 18 hours of data from 24 hour recordings the day after sauna treatment. Differences between these methods have been suggested, and with short-term recordings, HRV has been shown to return to baseline shortly after transient perturbations such as mild exercise (Camm et al, 1996). Furthermore, some research indicates 24-hour recordings are stable and ideal to assess intervention therapies (Camm et al., 1996). As such, it is possible that

the rest period between completion of sauna treatment and the final ECG acquisition, coupled with the short-term recording may account for the different findings in this study. It is important to note, however, that transient changes in some physiological measure can last for a day after perturbation. As such, it was necessary in the present study to perform final testing after a 48 hour “wash-out” period to ensure we could be observing adaptations and not transient responses.

Lastly, unlike previous research into the effects of sauna therapy (Kihara et al., 2002; Kihara et al., 2004; Imamura et al., 2001), we used a dry sauna instead of a far-infrared sauna due to availability and it is difficult to determine whether we achieved a similar degree of heat stress since core temperature changes during and after sauna exposure were not reported in that study. In the present study, sauna exposure was sufficient to elevate heart rate to between 92 and 103 beats per minute. The maximal heart rate values obtained during the $\text{VO}_{2\text{peak}}$ test ranged from 159 to 189 beats per minute. Thus, passive heating resulted in a heart rate response similar to that of low intensity exercise. Given that our population was in good general health, it is possible that the stress of sauna bathing was not sufficient to influence HRV.

The degree of heat stress may also interact with the renin-angiotensin-aldosterone system (RAAS) to influence HRV. In CHF, poor perfusion to the kidneys stimulates renin release (Nicholls, Robertson, & Inagami, 2001; Zablocki & Sadoshima, 2010). ANGII (a hormone that promotes blood vessel constriction), which is produced through the conversion of renin, is involved in the cardiac remodeling that occurs in CHF, as TGF- β , a protein associated with structural remodeling, is a downstream target of ANGII

(Huntgeburth et al., 2011). Furthermore, ANGII inhibits vagal activity (Townend et al., 1995). Athletes tend to have greater vagal activity as a result of lower plasma renin, therefore less ANGII inhibition (Fagard et al., 1985). Since low blood pressure stimulates the secretion of renin seen in CHF, it is possible that physiological stressors such as passive heating that increase blood pressure may reduce this renin secretion and ultimately reduce vagal suppression by ANGII. Since our sample population was generally healthy, we suspect the blood pressure increases during sauna exposure did not reduce renin secretion and ANGII inhibition of vagal activity, as participants did not have the excessive renin secretion associated with CHF. Further, we used a 2-week intervention similar to what Kihara et al (2004) implemented for CHF patients. Again, considering our population was free of CVD, the duration of the sauna exposure may have been too short. In an animal study, for example, Ikeda et al (2001) reported temperature increases similar to the present study, however the intervention was 4 weeks in duration.

Based on the current literature, we predicted declines in resting systolic and diastolic blood pressure (Imamura et al., 2001; Kihara et al., 2002). As with the HRV measure, sample size and the timing of the final physiological testing session may account for the differences in our results. Further, Kihara et al (2002) and Imamura et al (2001) examined the effects of sauna treatment in New York Heart Association Class II and III CHF patients. These individuals have a limited ability to participate in regular physical activity, as fatigue and dyspnea often occur. Passive heating may replace exercise for these individuals as it provides a mild cardiovascular stress with minimal risk. Our population were middle-aged, without cardiovascular or metabolic disease and

were able to safely perform a maximal exercise test. Our participants also ranged from low to moderately recreationally active. Therefore, they likely accumulated more physical activity per week than those in previous studies. Given that exercise produces shear stress on the vasculature, it is possible that the shear forces generated during the daily sauna exposure were comparable to, or even milder than what occurred during exercise. In support of this, one may compare maximal heart rate during the $\text{VO}_{2\text{peak}}$ test to the heart rate response during sauna exposure. Maximal values ranged from 159 to 189 beats per minute, while sauna bathing resulted in maximal values between 92 and 103 beats per minute. Cardiac output, as a function of heart rate and stroke volume, may not have been sufficiently increased to generate larger shearing forces on the vasculature. Therefore, the shear forces may have been insufficient to influence NO activity.

The timing of the final measurement may have been a factor in the blood pressure results as well. Kihara et al (2002) measured blood pressure one day following cessation of sauna treatment. Our final testing occurred between 48-72 hours following the final sauna exposure, in order to eliminate the acute effects of sauna use. A single moderate intensity aerobic exercise session is sufficient to influence 24-hour ambulatory blood pressure (Ciolac et al., 2008; Forjaz et al., 2000). Given the similar cardiovascular response to exercise and passive heating, it was possible that blood pressure may have been altered within 24 hours of sauna exposure. Despite this, it is possible that sauna bathing has no effect on resting blood pressure. In line with our findings, Kihara et al (2004) found no significant differences in resting blood pressure following 2 weeks of sauna treatment, despite measuring blood pressure one day after the final sauna bout.

While exercise and passive heating elicit similar cardiovascular responses, the immediate post-exercise increase followed by a delayed reduction in blood glucose is primarily mediated by working skeletal muscle. Passive heating is not accompanied by large increase in skeletal muscle substrate demands, however, increased blood flow to skeletal muscle has been suggested to underlie improvements in fasting plasma glucose and insulin sensitivity among diabetics following regular hot tub exposure (Ciolac et al., 2008; Hooper, 1999). Both local and whole body heat stress are associated with increases in skeletal muscle blood flow (Heinonen et al., 2011; Pearson et al., 2011). As such, a single sauna bath may not be sufficient to influence fasting blood glucose (this was indicated in the separate group of young subjects; data not shown), however, improvements may occur with repeated passive heating. Following our 2-week sauna intervention, blood glucose levels remained unchanged or slightly elevated among 4 of the 5 participants. Interestingly, the participant with the highest baseline blood glucose level showed a 15 mg/dL reduction following sauna treatment. Imamura et al (2001) and Hooper (1999) found improved fasting plasma glucose and insulin sensitivity among patients with at least 1 coronary risk factor and Type II Diabetics. It may be that sauna treatment is an effective glucose lowering therapy for diabetics or non-diabetics with elevated blood glucose as opposed to those with low-normal blood glucose.

No changes were observed for high density lipoproteins (HDL), low density lipoproteins (LDL), total cholesterol (TC), or triglycerides (TRG). It is likely that the duration of sauna exposure and the degree of cardiovascular strain account for these findings. HDLs are typically increased with regular exercise. In endurance trained individuals, increased lipid transfer to HDLs and decreased HDL clearance by hepatic

lipase contribute to a greater HDL apoA-I half-life and cholesterol excretion (Kraus & Slentz, 2009; Rashid & Genest, 2007). The duration of exercise, supported by the findings in endurance trained individuals, has been suggested as the most important element of training for HDL increase (Kodama et al., 2007). As such, the 15-minute sauna bath, while sufficient to elicit a response similar to exercise, may not have been of sufficient duration to influence HDL concentrations. Furthermore, LDLs are generally not responsive to exercise (Kraus & Slentz, 2009), and we did not anticipate significant improvements following sauna bathing. Since HDL and LDL concentrations remained unchanged, TC and the LDL:HDL ratio were also unchanged.

Hematocrit levels increased among all participants following 2 weeks of sauna exposure. Generally, exercise-induced hypoxia stimulates erythropoiesis (Smith, Bleyer, Little, & Sane, 2003) by increasing erythropoietin release from the kidneys. As sauna bathing is a form of passive heating, oxygen extraction in skeletal muscle does not create the hypoxic condition required to stimulate erythropoiesis. It has been shown, however, that oxygen extraction by essential organs such as the brain is increased during passive heating (Fan et al., 2008; González-Alonso et al., 2004). In order to counter the effects of passive heating, skin blood flow increases to aid in evaporative cooling while blood flow to essential organs is maintained, resulting in greater cardiovascular strain. These effects, coupled with greater oxygen extraction by the brain and other essential organs may have stimulated erythropoiesis in a similar manner as exercise stress.

Plasma volume increases with endurance training, as the result of greater aldosterone action (Convertino, 1991). Interestingly, during exhaustive exercise in heat,

aldosterone exhibits a temperature-dependent increase up to 38.5°C (Wright, Selkirk, & McLellan, 2010). Therefore aldosterone would be expected to increase as a result of sauna exposure, as average tympanic temperature was 37.9°C. Despite this, we observed a decrease in plasma volume, which may be due to the cardiorespiratory fitness of our population. Wright et al (2010) noted that aldosterone was significantly higher in trained ($\text{VO}_{2\text{peak}} = 70 \pm 2 \text{ ml/kg/min}^{-1}$) compared to untrained ($\text{VO}_{2\text{peak}} = 50 \pm 1 \text{ ml/kg/min}^{-1}$) individuals. The average $\text{VO}_{2\text{peak}}$ of our participants was $20.76 \pm 7.42 \text{ ml/kg/min}^{-1}$. The sauna treatment was the equivalent of low-intensity exercise, and lasted only 15 minutes. As such, this stress was insufficient to improve aerobic fitness and increase aldosterone action. Further, hydration status at the time of measurement may have contributed to these results.

Lastly, despite being instructed not to make any changes to physical activity levels for the duration of the study, participants may have reduced exercise due to time constraints. Participation in the study required approximately 45 minutes, five times per week. Since participants had family and work responsibilities, the sauna session may have replaced some of the time set allotted for exercise. While we suspect the stress of passive heating is similar to exercise stress, heat stress may have been less intense and shorter duration than regular aerobic exercise sessions. Therefore, the possibility of detraining resulting in lower plasma volume and greater hematocrit should be considered.

It is likely that body weight and body fat percentage did not change due to the fact that our sample population was between normal and overweight (mean BMI 25.66 ± 1.21), not obese. Reductions in body weight and body fat have been reported following

similar sauna treatments, however, participants were obese (Biro, Masuda, Kihara, & Tei, 2003). When normal weight individuals underwent the same treatment, increased plasma ghrelin accompanied appetite increases (Biro et al., 2003). As participants did not report appetite loss or reduction prior to the onset of the study, plasma ghrelin was likely within normal ranges. Further, the weight loss among obese patients was not accompanied by increased plasma ghrelin. It is suggested that obese individuals have a reduced ghrelin sensitivity, or maximal suppression of ghrelin secretion preventing a response following sauna treatment (Biro et al., 2003) and indicating the need for further studies examining the mechanisms of sauna use and weight loss. Therefore, since our sample population was normal weight to overweight and did not report appetite abnormalities prior to sauna treatment, plasma ghrelin changes may not have occurred, and weight remained unchanged. Dietary changes must also be considered, as the study relied on self-report.

Unlike Hayasaka et al (2008) we found no differences in anxiety scores (state and trait) following 2 weeks of sauna use. The timing of measurement may account for this discrepancy, as Hayasaka et al (2008) measured anxiety before and after a single session of passive heating while we measured anxiety 48-72 hours following the final sauna bath of 2 weeks of sauna therapy. Exercise training has been shown to reduce state and trait anxiety in a healthy middle aged population (Blumenthal, Williams, Needels, & Wallace, 1982), and given the similarities between exercise and heat stress we predicted improvements in STAI scores. However, the intensity of physiological stress may have been too mild during the sauna exposure, as Blumenthal et al (1982) employed an exercise intervention at 70-85% of VO_{2peak} . The mechanisms underlying anxiety reductions following exercise include increases in serotonin, central norepinephrine

transmission and a greater concentration of endorphins (Helmich et al., 2010). β -endorphins increase at approximately 80% $\text{VO}_{2\text{peak}}$ (McMurray, Forsythe, Mar, & Hardy, 1987), therefore the intensity of heat stress in the present study may have been too mild to influence endorphin release and psychological outcomes.

In conclusion, acute sauna exposure elicits a physiological stress response similar to that observed with low intensity exercise. However, 2 weeks of daily sauna use with a concomitant tympanic temperature increase of 1-1.5°C was not associated with improvements in markers of cardiovascular, metabolic and psychological health. Given the results of previous research, it is likely that this mode and duration of therapy is sufficient to improve the health of diseased individuals, but does not appear to make impact already healthy middle-aged adults.

2.6 Limitations and Future Directions

Kihara et al (2002) recruited CHF patients for a 2-week sauna therapy intervention. We recruited relatively healthy middle-aged adults, but were unable to successfully obtain enough participants to satisfy power requirements. We aimed to recruit 20-25 participants based on sample size calculations with a small-moderate effect size at an alpha level of 0.05, and a power of 0.80. This was based on the data of Kihara et al (2004) who reported large effect sizes for HRV measures. Unfortunately, participant interest in the study was never strong and of those who were interested, several did not meet inclusion/exclusion criteria. As a further hindrance to our recruitment efforts, the saunas at the University of Windsor were opened only for males and then closed due to

unforeseen circumstances partly through the study. Because the power was so small, definitive conclusions could not be made with this dataset. However, in the small sample size in which data was collected, sauna therapy appeared to have no effect on markers of cardiovascular or psychological health.

Secondly, previous studies used far-infrared saunas and reported only that the saunas were maintained at 60°C temperature (Imamura et al., 2001; Kihara et al., 2002, 2004, 2009). In the present study, dry saunas were used which were located in fitness centres, resulting in inconsistent temperatures as the general public were able to access the sauna during testing, allowing warm air to exit periodically throughout sauna bathing. Although this field study may have resulted in a milder heat stress compared to a laboratory setting, these observations would be what the general public would experience if attending either of the fitness facilities incorporated in this study. If the health benefits of sauna treatment are going to be purported by fitness centres, it is imperative that the knowledge of the therapeutic parameters of a sauna be conveyed to its staff and members. Moreover, future studies should examine the effects of greater heat stress or a longer duration sauna exposure, either as a longer bout of passive heating or more long-term use. Greater heat stress may be associated with improvements in health markers.

Participants were also instructed not to alter their diet or exercise habits for the duration of the study, however it is possible that this was difficult to maintain, as a portion of the study occurred during the winter holidays when routines may have been difficult to uphold. Dietary and activity records were not kept during this period and it is possible that this had an effect on the ability to detect physiological changes.

Finally, in an effort to account for HRV fluctuations during the menstrual cycle (Bai et al., 2009; Baker et al., 2008), baseline values from the initial visit were compared to post-sauna values. However, one participant experienced amenorrhea in the months leading up to the study. The amenorrhea was a menstrual cycle irregularity, however the participant was not considered fully menopausal. Thus, pre-sauna and post-treatment values were compared. Nonetheless, while it is likely that the length of time between initial and post sauna testing would have put the female individuals in the same phase of the menstrual cycle, this is unknown. However, given that there were no significant differences observed in any measure of HRV suggest that even if there were changes, these would not be profound adaptations because menstrual cycle could override them. Work in this area needs further investigation.

Despite these limitations, the outcomes of this study could provide insight into novel strategies to prevent the onset of cardiovascular and metabolic disease. Passive heating was able to significantly alter the physiology (temperature and HR) of healthy young and middle-aged adults. However, the physiological stresses of 2 weeks of 15 min sauna baths may not be great enough to elicit lasting adaptations. Future studies should examine greater increases in core temperature and/or greater durations of heat acclimation in these populations in order to determine their therapeutic use. Currently, adherence rates to traditional physical activity programs are low. Thus, the development of new strategies requiring minimal effort and time commitment may improve adherence and improve health markers in the middle-aged population, ultimately reducing the incidence of cardiovascular and metabolic disease-related deaths.

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Appendices

Appendix A: Physical Activity Readiness Questionnaire (PAR-Q)

Physical Activity Readiness
Questionnaire - PAR-Q
(revised 2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
<input type="checkbox"/>	<input type="checkbox"/>	1. Has your doctor ever said that you have a heart condition <u>and</u> that you should only do physical activity recommended by a doctor?
<input type="checkbox"/>	<input type="checkbox"/>	2. Do you feel pain in your chest when you do physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	3. In the past month, have you had chest pain when you were not doing physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
<input type="checkbox"/>	<input type="checkbox"/>	5. Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
<input type="checkbox"/>	<input type="checkbox"/>	7. Do you know of <u>any other reason</u> why you should not do physical activity?

If
you
answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

- If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:
- start becoming much more physically active — begin slowly and build up gradually. This is the safest and easiest way to go.
 - take part in a fitness appraisal — this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

DELAY BECOMING MUCH MORE ACTIVE:

- if you are not feeling well because of a temporary illness such as a cold or a fever — wait until you feel better; or
- if you are or may be pregnant — talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME _____

SIGNATURE _____

DATE _____

SIGNATURE OF PARENT
or GUARDIAN (for participants under the age of majority) _____

WITNESS _____

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.



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continued on other side...

Appendix B: Participant Information Sheet

Name: _____

D.O.B. (dd/mm/yy) ____/____/____

Sex: m____ f____ prefer not to disclose _____

Last start date of menstrual cycle (females only): _____

Contact information:

Phone: () _____ - _____

E-mail: _____ @ _____

Emergency contact (optional)

Name: _____

Phone: () _____ - _____

Appendix C: Medications/Medical Conditions

Are you experiencing or have you experienced any of the following conditions (check all that apply):

- Aortic stenosis (valvular heart disease) [☐]
- Unstable angina pectoris [☐]
- Myocardial infarction [☐]
- Heart failure [☐]
- Cardiac arrhythmia [☐]
- Hypertension [☐]
- Hypotension [☐]
- Orthopedic conditions (ie. joint pain, limited mobility) [☐]
- Are you currently pregnant? [☐]
- Do you have any of the following cardiovascular disease risk factors?
 - Family history of heart disease [☐]
 - Smoking [☐]
 - Sedentary lifestyle (less than 30 min of moderate intensity exercise 3 days per week) [☐]
 - Obesity [☐]
 - Hypertension (blood pressure greater than 140mmHg/90mmHg, or diagnosed by a physician) [☐]
 - Dyslipidemia [☐]
 - Diabetes or prediabetes [☐]

- Are you taking any of the following medications?
 - Diuretics (used to treat high blood pressure)
 - Anticholinergics (ie. Cogentin, Atropine, Atrovent, Spiriva, Elavil, Tofranil, and Anafranil)
 - Barbiturates (ie. Seconal, Nembutal, Amytal, and Tuinal)
 - Beta blockers (ie. Sectral, Tenormin, Kerlone, Lopressor, etc)
 - Antihistamines

[] Yes, I am currently taking one or more of these medications.

[] No, I am not taking any of these medications.

Name: _____

Date (dd/mm/yy) ____/____/____

Signature:_____

Appendix D: State Trait Anxiety Inventory (STAI)

Due to the terms of use of the STAI (see next page), a sample of only 5 questions may be used in this document.

Sample Items

I feel calm.....1

2 3 4

I am tense.....1

2 3 4

I feel self confident.....1

2 3 4

I feel nervous.....1

2 3 4

I am relaxed.....1

2 3 4



www.mindgarden.com

To whom it may concern,

This letter is to grant permission for the above named person to use the following copyright material;

Instrument: ***State-Trait Anxiety Inventory for Adults***

Authors: ***Charles D. Spielberger, in collaboration with R.L. Gorsuch, G.A. Jacobs, R. Lushene, and P.R. Vagg***

Copyright: ***1968, 1977 by Charles D. Spielberger***

for his/her thesis research.

Five sample items from this instrument may be reproduced for inclusion in a proposal, thesis, or dissertation.

The entire instrument may not be included or reproduced at any time in any other published material.

Sincerely,

Robert Most
Mind Garden, Inc.
www.mindgarden.com

Appendix E: Ethics Clearance



Office of the Research Ethics Board



Today's Date: November 04, 2011
Principal Investigator: Ms. Michelle Dotzert
REB Number: 29438
Research Project Title: REB# 11-167: The Effects of Sauna Bathing on Health Markers in Middle Aged Males and Females
Clearance Date: November 4, 2011
Project End Date: September 30, 2012
Milestones:
Renewal Due-2012/09/30(Pending)

This is to inform you that the University of Windsor Research Ethics Board (REB), which is organized and operated according to the Tri-Council Policy Statement and the University of Windsor Guidelines for Research Involving Human Subjects, has granted approval to your research project on the date noted above. This approval is valid only until the Project End Date.

A Progress Report or Final Report is due by the date noted above. The REB may ask for monitoring information at some time during the projects approval period.

During the course of the research, no deviations from, or changes to, the protocol or consent form may be initiated without prior written approval from the REB. Minor change(s) in ongoing studies will be considered when submitted on the Request to Revise form.

Investigators must also report promptly to the REB:

- a) changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
- b) all adverse and unexpected experiences or events that are both serious and unexpected;
- c) new information that may adversely affect the safety of the subjects or the conduct of the study.

Forms for submissions, notifications, or changes are available on the REB website: www.uwindsor.ca/reb. If your data is going to be used for another project, it is necessary to submit another application to the REB. We wish you every success in your research.

Pierre Boulos, Ph.D.
Chair, Research Ethics Board

c.c. Dr. Kevin Milne, Supervisor, Kinesiology

This is an official document. Please retain the original in your files.



Vita Auctoris

Michelle was born in Windsor, Ontario in 1988 and completed her undergraduate and graduate degrees in Kinesiology at the University of Windsor. She is passionate about physical activity and hopes to pursue a career in health research or medicine.